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RECENT ADVANCES IN THE SURGICAL TREATMENT OF FACIAL PARALYSIS AND BELL'S PALSY.*

JOSEPH A. SULLIVAN, M.B.,
Toronto, Canada.

Otology owes a great debt to the late Sir Charles Ballance, and the late Dr. Arthur Duel, in establishing a definite pattern for surgical interference in this gross and appalling deformity, facial paralysis. As a matter of fact, to these men should be given credit for laying the modern foundation of meticulate temporal bone surgery under magnification, which was later followed by the high speed drill technique.

The small amount of pathological material available to illustrate the causes and nature of the various lesions responsible for peripheral facial paralysis is partially due to the essentially harmless nature of the condition. Moreover, in those cases in which the injury, disease or growth responsible for the paralysis have spread to neighboring and more vital structures with fatal results, the florid picture presented by the facial condition has overshadowed the facial paralysis with the result that the opportunity to collect material for detail examination has been overlooked.

Some cases of facial paralysis that have been seen recover spontaneously and this has in the past been responsible for the custom of relying upon any inherent powers of recuperation with which the nerve appears to have been endowed.

*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Atlanta, Ga., Jan. 13, 1952.

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The bony facial canal is an integral part of the pars petrosa throughout its entire course. This applies to its embryological development and to its surgical anatomy; therefore, it may be said that it is closely related to pathological changes occurring within the temporal bone. It is an established fact that the cellular structure of the temporal bone varies greatly. This equally applies to its constituent bony facial canal, the internal aspect of which is the anatomical pathway to the bony area of the bulb and the inferior surface of the petrosal pyramid. The cellular character of the canal, varying in direct relation to the progress of invasion from the antrum and the middle ear, is normal or modified by early pathological change. Cellular extension from the antrum seldom occurs prior to the second year of life. Until that time the normal histological appearance may be considered to be diploic.

ANATOMY.

It is essential that we trace the course of the facial nerve fibres from the cortex to their distribution to the muscles of the face in order to be able to localize with any certainty the position of a lesion causing facial paralysis. It must be kept in mind, however, that it is only surgically possible to combat lesions of this structure which occur in the Fallopian canal.

The motor fibres of the VIIth nerve to the facial musculature arise in the pons in a cell column common to all the motor cranial nerves except the hypoglossal nerve and nerves to the eye. This facial nucleus included in the basal reflex arc is controlled from the cortex in a manner that has special clinical significance. While the corticobulbar tract reaches it undivided, there is a separation of the higher centres controlling each nucleus, so that the temporal fibres for the occipitofrontalis, orbicularis oculi and corrugator supercilli are not involved in cortical hemiplegia. Starting from the cortex, the motor fibres run down to the seventh nucleus, situated deep in the lower portion of the pons, and they emerge as the motor root at the lower border of the pons, between the olive and restiform body. Just before the motor root leaves the pons it is joined by fibres from the third nucleus,

which supply the following muscles: the orbicularis palpebrarum, corrugator supercilli and the anterior belly of the occipitofrontalis; therefore, lesions occurring in the cortex, or in the course of the fibres to the nucleus, or a little beyond will cause paralysis of the muscles of the face with the exception of these three; a questionable deafness due to paralysis of the stapedius muscle, and no loss of taste in the anterior two-thirds of the tongue. There will be no dryness of the mouth, as the fibre supply of vasodilator nerve impulses to the submaxillary and sublingual glands are not involved. The electrical reactions will be normal. The lesion may be traumatic, embolic, or due to a new growth, and usually entails paralysis of the VIth nerve and hemiplegia. The diagnosis of a lesion in this area is by the fact of the escape of the three muscles mentioned.

Proceeding further along the course of the motor root, the next part of importance is the internal auditory meatus. Occasionally the nerve is injured in the removal of an acoustic tumor. Conversely, the sensory root, which is closer to the VIIIth nerve than the motor root, may be injured. In the internal meatus, that is, up to the geniculate ganglion, injury is most commonly due to fracture of the base of the skull, and the VIth, VIIth and VIIIth nerves are usually involved.

The next important diagnostic site from the surgical point of view is from the geniculate ganglion to the stylomastoid foramen, which is the Fallopian aqueduct. In the lower portion, that is, the vertical segment, which is between one-half and three-quarters of an inch long, the nerve runs through dense bone, and it is in this portion only that Bell's palsy occurs. In my opinion, the chorda tympani nerve is the only important anatomical branching from the facial nerve that allows us to localize accurately the site of the lesion, and this is done by testing for the sense of taste on the anterior two-thirds of the tongue. Lesions of the nerve after it leaves the stylomastoid foramen are usually traumatic during operation or accident, or due to new growth of the parotid gland and are easily distinguishable. From an otological standpoint the

facial nerve traverses three separate routes: the labyrinthine, 4 mm.; the tympanic, 8 mm.; and the mastoid segment, 7 mm.

The labyrinthine segment enters the bone through the internal auditory meatus from the posterior cranial fossa, running from within outward in the horizontal plane between the cochlea and the vestibule, ending in the geniculate ganglion. The nerve in this location is not accessible to injury during a mastoid operation. It then makes a right angled turn at the genu, commencing the tympanic segment, which enters the Fallopian canal on the internal wall of the middle ear just above and slightly anterior to the point where the ligament of the tensor tympani muscle is given off for its attachment to the handle of the malleus. This ligament is a reliable guide to the position of the nerve during the radical operation. The tympanic segment runs directly backward and slightly outward through the Fallopian canal in the horizontal plane until it passes between the horizontal semicircular canal and the oval window; then it makes another right angled turn, forming the mastoid segment, which runs directly downward in the vertical plane along the bottom of the posterior canal wall, leaving the temporal bone through the stylomastoid foramen at the mastoid tip. In its horizontal course across the internal wall of the middle ear through the Fallopian canal the nerve is covered by an extremely thin layer of bone and is directly accessible to injury by the curette or undue pressure during the radical operation. The average length of the facial canal in the adult is about 24 mm. The bend between the mastoid and tympanic segments is termed the pryamidal segment and its average length is from 2 to 6 mm.

The facial canal is a compact structure, varying in length and diameter. Its average diameter in its mastoid segment is 4 mm.; tympanic segment, 2.5 mm.; at the stylomastoid foramen, 4 to 6 mm. The contents of the canal, that is, the sheath, nerve and vascular supply, account for one-third or more of its transverse diameter. It has been erroneously stated that in the upper portion these structures were more compactly placed than in the lower portion or at the stylomastoid fora-

men. I cannot accept this statement, because according to our investigations the facial nerve just within and at its exit from the stylomastoid foramen is so compactly bound down and intimately surrounded by a dense periosteal sheath that it has less space to expand than if it were encased by the bony walls of the aqueduct, which canal it does not completely fill. I consider this finding amply substantiated in the clinical manifestations of the commonly prevalent peripheral type of so-called Bell's palsy.

Our observations have revealed, in their order of importance, that the following anatomical sites are the most commonly involved in surgical instrumentation in the temporal bone resulting in facial paralysis:

1. The creation of an artificial antrum below and distal to the horizontal canal.

2. Failure to identify the tympanic portion of the nerve proximal to the horizontal canal in the radical operation.

3. Injury to the vertical segment of the nerve in an extensively pneumatized bone in removal of the retrofacial group of cells.

4. Injury to what is called the pyramidal segment of the nerve in the planing down of the hypotympanum on the posttympanic wall.

5. The least common, either in infants or adults, injury to the nerve in the soft parts after its exit from the stylomastoid foramen excluding, of course, injuries and tumors.

In a previous writing, we stated that there are certain maxims that should be followed. Time does not allow me to go into each one of these in detail, but I wish to speak about one.

That part of the nerve which occupies the canal between its tympanic and mastoid portions is the pyramidal segment. I wish to emphasize this region, which I call the pyramidovertical or pyramidomastoidal segment. It is much more vulnerable, not in the lowering of the posterior bony canal (or

facial ridge) or knocking off the so-called facial spur, but on its inner aspect, the posterior tympanic wall region which must be subjected to thorough and accurate curettage in the removal of the annulus tympanicus and the requisite planing down of the hypotympanum, a fundamental step in the carefully executed complete operation. This also is a danger area, in the fenestration operation in the removal of the bone, over and anterior to the posterior end of the horizontal canal, just below the chorda tympani nerve.

It is advisable at the ouset in our consideration of the various methods of repair to state frankly that the recovery after the union of one nerve with another and even after direct suture of a divided nerve is never functionally perfect, although often amazingly satisfactory when compared with the preceding paralysis. We should strive for two muscular actions: the ability to close the eye and the ability to elevate the corner of the mouth. Generally speaking, paralysis of all the muscles supplied by the facial nerve, combined with an inability of the nerve to conduct a faradic current applied through the skin, but with a response to galvanic stimulation of the musculature, is an indication of a lesion of the nerve fibres. To determine which cases are suitable for operation and which are not, I rely not only upon the patient's reaction to serial faradic interpretations but also upon my experience gained over a period of years in the observations of these patients. I particularly stress the importance of magnification in any procedure on the facial nerve. I have been using it now for a period of 20 years. Not only magnification but also a dissecting microscope giving 14 diopters of magnification, and it has been since the use of the latter that I was drawn to enunciate certain ideas, both in relation to nerve grafting, suturing and the plasma technique which previously had not been apparent under ordinary vision. Along with Tickle and Cawthorne, the importance of the electrical reaction is stressed. I have always made it a point to see for myself these reactions are carried out; particularly in the use of the faradic current. It is often necessary to expose the nerve directly at the stylomastoid foramen; there will

not then be any question that the electrode is placed directly on the nerve, eliminating the spread effect on adjacent muscles.

ELECTRICAL REACTIONS.

Muscle atrophy and the resulting fibrotic changes are soon apparent in the thin musculature of the face where there is no definite muscle fascia surrounding the individual muscles. These are inserted into subcutaneous tissue or skin without the intervention of any subcutaneous fascia. When denervation has persisted for any length of time the fibrotic sheath which replaces the individual muscle cannot be made to contract even if continuity of the nerve could be restored. The importance of physical treatment of the facial muscles in every patient with VIIth nerve palsy cannot be overestimated. The best surgical technique which will restore conduction of the nerve impulse is useless if the facial muscles have been allowed to stretch and atrophy before the operation or while nerve regeneration is taking place.

FARADISM.

A normal muscle responds to percutaneous faradic stimulation at low intensities. If the nerve degenerates, its excitability decreases and is ultimately lost. It is taken for granted that we realize that faradism is a direct stimulation of short duration to nerves. The muscle contracts because it is receiving impulses via the intramuscular nerve twigs. Galvanism is a direct stimulation to muscle tissue. The galvanic response is much more easily elicited. Failure to elicit a galvanic response in muscle in the absence of edema indicates serious atrophic change in a muscle. Fibrous tissue seems to wage a war of attrition upon muscle fibres.

ELECTROMYOGRAPHY.

From its history it is obvious that electromyography as it is known today is a direct application of electrophysiology to clinical medicine. The action current of contracting muscles can be recorded. The electrocardiogram is a well known ex-

ample. By electromyography it is possible to record the action current in a voluntary muscle. After section or severe compression of a nerve the muscle fibres supplied by it naturally lose their power of contracting on volition as well as the collective electrical disturbances associated with this. It does not, however, spell the end of activity of the individual muscle fibres composing the motor units. After a lapse of approximately three weeks these fibres may begin to enjoy an autonomous life, of which one manifestation is a rhythmic activity termed "fibrillation." As a result of muscle study, it is revealed that surgical repair, if necessary, should be undertaken as soon as possible. In our experience the interpretation of electromyography results is strictly limited in facial palsy, and will show only a few active fibres, two to four weeks after the onset of the lesion, when damage already has been done.

Weddell states that the most difficult muscles to relax are those of the face, tongue and abdomen. The presence of fibrillation is considered of great diagnostic and prognostic significance in peripheral nerve injury; however, there are other symptoms in differentiating quantitatively between complete and partial denervation and qualitatively between paralysis due to transient nerve block and that due to axon degeneration. At the moment, electromyography may well be possible in the future to standardize recording technique in terminology to such an extent as to make this practicable, but the time has not yet come; furthermore, the clinician, regardless of the findings by this method, can confirm or reject a tentative diagnosis either on clinical grounds or as a result of an exploration.

In a case of inexcitability of a nerve below the level of the lesion it does seem probable that the retention of the faradic response is related to the presence of a few undegenerated nerve fibres.

In some cases of facial paralysis it is difficult to obtain an accurate result by the faradic test; this is due to an unduly high skin resistance. The faradic response may not be ob-

tained in this case, yet when the nerve is exposed and an electrode applied directly to it, conductivity can be demonstrated. For this reason the nerve should be exposed and stimulated in all questionable cases; another finding that lends itself to this important prerequisite is the very fact that pain is a factor in faradic stimulation and as I have previously stated on many occasions, this is why, in order to be of a degree of finality, the test should be carried out under an anesthetic. Weddell states, "It is not possible by electromyography to distinguish between a total axonal interruption and a complete ischemic block for some days after the injury." Weddell's observations have not included many cases of traumatic facial paralysis or Bell's palsy where their findings for a great majority of these cases were quite unspecific. He further states that ischemic block may persist in unlimited periods and in such a case recovery might be expected to follow decompression, unless a persisting infection is present. There is experimental and clinical evidence that atrophy of muscles can be retarded by activity induced by adequate electrotherapy. The contraction of the muscle is essential; therefore, faradic stimulation of denervated muscle is useless and a waste of time. Denervated muscle responds to galvanism and this is one method of treatment.

A detailed method of exposure of the nerve has been fully described by the author in a previous writing.

NERVE GRAFTS.

It is now an accepted principle in neurosurgery that a gap in an injured nerve can be bridged by means of a nerve graft. A controversial point has arisen whether to use a fresh or degenerated graft. Bently and Hill, writing in the *British Journal of Surgery*, clearly describe an important piece of research work in this particular regard. In conjunction with Dr. Keith, of the Department of Surgery at the Banting Institute of the University of Toronto, we carried out investigations in nerve degeneration and regeneration in an attempt to demonstrate that the neurofibrils grew through the empty tubules. Our experimental results proved that there was sat-

isfactory clinical evidence of regeneration through the graft, after removal and histological examination. The clinical results in patients amply substantiate this finding. This has been recorded in previous publications.

BELL'S PALSY.

The following is a summary of a paper read by the author before the American Otological Society, San Francisco, May, 1950. It is suggested that the reader refer to that paper for the details in full.

The damaging effect of pressure on a nerve is due to occlusion of the blood vessel. This dysregulation of the circulation, as I prefer to call it, affects primarily the myelin sheath and has little if any influence on the Schwann cells and axis cylinders. Here is the temporary block to conduction, but continuous pressure produces other changes. Vascular stasis occurs in neighboring segments, leading to edema above and below the site of compression, as we have shown. Not only is the process of repair in the compressed area impeded, but a complete degenerative lesion involving the axis cylinders and Schwann cells is finally produced. It is thus seen how reversible ischemic block, which may be present for lengthy periods in nerves of the limbs without leading to degeneration, will by persisting in the rigid facial canal produce increased vascular stasis and edema leading to complete degeneration. Pain accompanying complete facial paralysis is indicative of a severe degenerative lesion.

What is the fundamental neurosurgical principle involved in crushing injuries involving nerves and bones anywhere in the body but that of relieving the pressure on the involved nerve, thus mobilizing the nerve and allowing it to regain its functional condition as soon as possible. If that is the principle involved in soft tissues, what must it be of a motor nerve encased in a bony canal? The extreme degree of edematous swelling which immediately prevails with marked swelling of the nerve bundles becoming manifest after the careful removal of the upper two-thirds of the bony canal without

damage to the blood supply as far as it is surgically possible, and incising the epineural connective sheath along the side of the nerve instead of on its upper aspect in order to prevent damage to the longitudinal arterial anastomosis has been revealing to me over a period of 20 years in the examination of this nerve under a dissecting microscope. This appearance has been observed anywhere from three to 12 weeks, and particularly above the area of the actual foramen itself with less manifestation of this macroscopic change as the nerve extends outward and divides at the pes anserinus. The rapidity of recovery following this procedure is only too self-evident.

The appearance of the nerve in cases anywhere from six months to a year following this change in which all that has remained has been a fibrous strand more than anything else vindicates the rationale of this procedure.

Merwarth states that recurrences of facial paralysis are fairly common, and particularly is this true in Bell's palsy. It is, therefore, interesting to recall the fact that in all my cases subjected to surgical intervention no recurrence of facial paralysis has resulted on the operated side; similarly, that finding is true in the cases of Cawthorne, of England. This means that compression, in addition to the dysregulation of the circulation, is the cause or the factor in the production of this lesion in the VIIth nerve.

We are dealing with a cranial nerve, a motor nerve, which has to do with muscular power and emotional expression, with facial muscles lacking a fascial covering and prone to rapid muscular atrophy. If the triad of symptoms I have just enunciated is present, you cannot decompress the facial nerve too early.

Some claim that vasospastic states can produce changes in nerves. This applies much more to a sensory nerve, such as the Vth nerve, than it does to a motor nerve, such as the facial nerve. For example, in patients with severe Vth nerve neuralgia the administration of a vasodilator drug in some cases has helped. There is also the possibility of involvement

of the VIIIth cranial nerve and its end-organ in the typical condition of hydrops of the internal ear, but this is vasospasm within the Vth or the VIIIth nerve at the root of a sensory disturbance. The argument which has been presented in the latter has been based upon examination and experience with sensory nerves. I do not feel that this applies to the pure motor facial nerve.

TO SUMMARIZE:

1. Facial paralysis immediately following a mastoid operation, accompanied by loss of the faradic current, demands exploration.

2. Where the nerve-ends can be placed in apposition within the Fallopian aqueduct, without tension, this is the procedure of choice.

3. Where the intervening space as a result of the injury requires the interposition of a graft of less than 8 mm., a fresh graft may be used.

4. Where the intervening space is 8 mm. up to 30 mm., a degenerated graft is preferable for the reasons given.

5. The rerouting of the nerve is not advised.

6. The graft should be held in position by means of some retention substance, and for this procedure the use of the plasma technique is advocated.

7. A Bell's palsy presenting complete facial paralysis, loss of the faradic response accompanied by pain, is indicative of a severe degenerative lesion and warrants immediate surgical interference.

CORRELATION OF THE PREDICTED WITH THE ACTUAL RESULT OF FENESTRATION IN 164 CONSECUTIVE CASES.*†

GEORGE E. SHAMBAUGH, JR., M.D.,
Chicago, Ill.

Two uncertainties that confront the patient who is contemplating the fenestration operation are the immediate hearing result that he will attain and the permanence of this result.

In an attempt to diminish the first of these uncertainties, a simple, practical and dependable method was sought for predicting within a few decibels the level of hearing that might be anticipated from surgery in a particular patient. In September, 1950, a simple formula for predicting the probable result of fenestration was first described.[1] This formula is based upon two propositions: That the bone conduction audiogram when made with proper precautions and after certain corrections is a reliable and accurate measure of cochlear reserve; that the successfully fenestrated ear has a certain residue of unrestored conductive loss due to the altered mechanics of sound conduction.

THE BONE CONDUCTION AUDIOGRAM IN OTOSCLEROSIS.

Many otologists distrust bone conduction audiometry, feeling that its results are sufficiently variable and unreliable to have little practical value. Carhart[2] has pointed out the reasons for this distrust and has suggested practical remedies for the practicing otolaryngologist. These include proper calibration of the bone unit to give superimposed air and bone

*Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Inc., Indianapolis, Ind., Jan. 28, 1952.

†From the Department of Otolaryngology, Northwestern University Medical School.

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curves in normal ears and in pure perceptive losses; correct audiometric technique in sufficiently quiet, sound treated testing rooms, and the use of proper masking.

By a careful statistical analysis of test-retest results of air as compared with bone audiometry, Carhart and Hayes[3] demonstrated that *when appropriate precautions are observed the otolaryngologist may accept with equal confidence the precision of his bone conduction and air conduction audiometric measurements.*

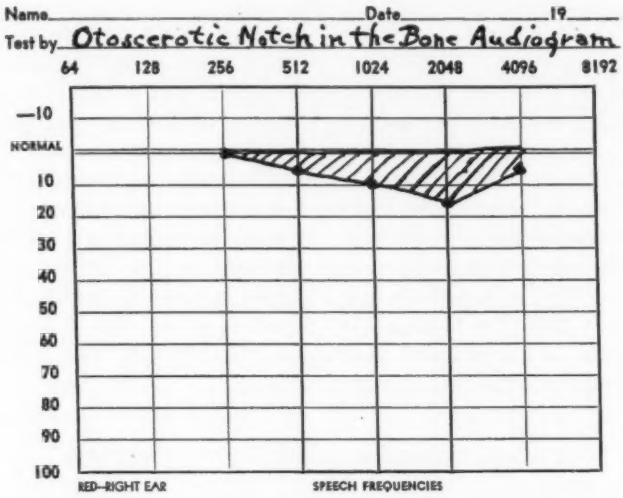


Fig. 1. The average correction in the bone conduction audiogram of otosclerosis, according to Carhart, required to determine the true cochlear reserve.

Juers[4] was the first to call attention to a fairly consistent improvement in bone conducted hearing following fenestration, greatest for the 2,048 frequency. Carhart[1] subsequently pointed out that stapes ankylosis produces a characteristic modification of the bone conduction audiogram consisting of a shallow notch that includes the frequencies of 512 through 4,096, deepest at 2,048. Since this notch largely disappears after fenestration and it is unreasonable to believe that fenes-

tration reverses or removes cochlear nerve degeneration, Carhart suggests that it is a mechanical effect of stapes fixation. To determine the true cochlear reserve of a case of clinical otosclerosis the bone conduction audiogram needs to be corrected for this mechanical notch. While the notch probably varies in depth in different ears, it averages about 5 db for 512, 10 db for 1,024, 15 db for 2,048, and 5 db for 4,096 (see Fig. 1).

The bone curve corrected for the Carhart notch may be taken as the measure of the cochlear reserve.

THE RESIDUE OF UNRESTORED CONDUCTIVE LOSS AFTER FENESTRATION.

The normal ear possesses a lever-mechanism to overcome the difference in sound conduction in air as compared with water. This mechanism consists of a large tympanic membrane, an ossicular chain and a small oval window to convert air vibrations of large amplitude and small force into water vibrations of small amplitude and large force. Deprived of this "impedence-matching" lever mechanism, an ear loses between 20 and 30 db of hearing acuity, according to various estimates. While the mechanics of the fenestrated ear are not well understood, it is clear that it does not possess an impedence-matching lever mechanism, so that we should expect a residue of unrestored conductive loss after fenestration of between 20 and 30 db. Davis and Walsh[5] found that a conductive deficit of 20 db was the *upper* limit of improvement in a group of operated cases without cochlear degeneration. Tentatively, the author has selected 25 db as the *average* residue of unrestored conductive loss after a successful fenestration.

FORMULA FOR PREDICTING THE RESULT OF FENESTRATION.

To lessen errors inherent in audiometry, three bone and air audiograms are made on different days preoperatively in quiet, inside sound-treated testing rooms on instruments calibrated to give a zero reading for both air and bone on normal ears. The speech frequencies in the three bone audiograms

CASE	FENESTRATION AND SIDE	DATE	AVERAGE PRE-OP LEVEL FOR 512, 1024, 2048 AC	BC	ESTIMATE OF POST-OP RESULT	ACTUAL 6 MOS. RESULT	DISCREPANCY BETWEEN ESTIMATE & ACTUAL	GAIN OR LOSS IN db AFTER FEN.	UNOPER. EAR.
A	2747 L	10-16-50	52	13	28	28	0	+24	+1
A	2748 R	10-16-50	46	12	27	28	-1	+18	-1
A	2749 R	10-17-50	64	11	26	23	+3	+41	-3
A	2750 L	10-17-50	50	11	26	25	+1	+25	+4
A	2751 L	10-17-50	43	1	16	32	-16	+11	-5
A	2752 R	10-18-50	47	10	25	28	-3	+19	+6
B	2753 L	10-18-50	62	21	36	43	-7	+19	+1
A	2754 R	10-19-50	47	9	24	25	-1	+22	-6
B	2755 L	10-20-50	46	21	36	28	+8	+18	-3
A	2756 L	10-20-50	54	11	26	35	-9	+19	0
A	2757 L	10-23-50	47	8	23	17	+6	+30	+8
A	2758 L	10-23-50	55	7	22	27	-5	+28	-5
C	2759 L	10-24-50	79	27	42	42	0	+37	+2
A	2760 L	10-24-50	46	8	23	25	-2	+21	0
B	2761 L	10-24-50	53	13	28	25	+3	+28	+10
A	2762 R	10-25-50	55	5	20	38	-18	+17	-7
A	2764 R	10-25-50	43	12	27	37	-10	+6	-4
A	2765 R	10-26-50	55	9	24	40	-16	+15	+7
A	2766 R	10-26-50	40	0	15	25	-10	+15	-1
B	2767 L	10-27-50	64	14	29	30	-1	+34	+2
A	2769 R	10-27-50	60	9	24	32	-8	+28	+2
A	2770 L	10-31-50	46	13	28	27	+1	+19	+1
A	2771 R	11-2-50	49	12	27	22	+5	+27	+3
A	2772 L	11-6-50	48	13	28	28	0	+20	-2
A	2773 R	11-7-50	48	2	17	22	-5	+26	-5
A	2774 L	11-9-50	45	9	24	30	-6	+15	-1
B	2775 L	11-10-50	51	23	38	35	+3	+16	+6
A	2776 R	11-10-50	42	7	22	20	+2	+22	-1
A	2777 L	11-10-50	42	11	26	35	-9	+7	+1
B	2778 R	11-10-50	58	21	36	38	-2	+20	0
B	2779 R	11-11-50	52	22	37	37	0	+15	-1
A	2780 L	11-13-50	53	7	22	23	-1	+30	-9
A	2781 R	11-14-50	57	13	28	30	-2	+27	+3
A	2782 R	11-14-50	67	7	22	27	-5	+40	+5
A	2783 L	11-16-50	47	14	29	18	+11	+29	+6
B	2784 L	11-17-50	57	17	32	43	-11	+14	+2
C	2785 R	11-17-50	77	32	47	37	+10	+40	+3
B	2786 L	11-17-50	61	21	36	43	-7	+18	+3
A	2787 L	11-17-50	52	11	26	20	+6	+32	+3
A	2788 R	11-20-50	55	6	21	25	-4	+30	-3
A	2789 R	11-20-50	36	13	28	25	+3	+11	+6

CASE	FENESTRATION AND SIDE		DATE	AVERAGE PRE-OP LEVEL FOR 512, 1024, 2048 AC	BC	ESTIMATE OF POST-OP RESULT	ACTUAL 6 MOS. RESULT	DISCREPACNY BETWEEN ESTIMATE & ACTUAL	GAIN OR LOSS IN db AFTER FEN.	UNOPER. EAR.
A	2790	L	11-20-50	49	13	28	32	-4	+17	+4
C	2791	R	11-7-50	70	30	45	42	+3	+28	+7
A	2792	R	11-24-50	48	8	23	22	+1	+26	+7
A	2793	L	11-27-50	54	15	30	33	-3	+21	-1
A	2794	R	11-27-50	52	11	26	28	-2	+24	-5
A	2795	L	11-27-50	54	8	23	40	-17	+14	-1
B	2796	R	11-28-50	68	19	34	58	-24	+10	+5
A	2797	R	11-28-50	50	10	25	27	-2	+23	-2
A	2798	R	11-30-50	43	10	25	33	-8	+10	-5
A	2799	L	12-1-50	54	11	26	22	+4	+32	0
B	2800	L	12-1-50	59	15	30	26	+4	+33	-4
A	2801	R	12-1-50	48	8	23	22	+1	+26	+1
C	2802	L	12-4-50	71	35	50	52	-2	+19	+1
A	2803	L	12-4-50	49	15	30	23	+7	+26	-2
B	2804	L	12-5-50	54	19	34	47	-13	+7	+2
A	2805	R	12-5-50	54	14	29	23	+6	+31	+5
B	2806	R	12-7-50	67	24	39	40	-1	+27	-1
A	2807	L	12-7-50	57	8	23	27	-4	+30	-3
A	2808	R	12-8-50	62	13	28	30	-2	+32	0
B	2809	L	12-11-50	55	17	32	28	+4	+27	0
B	2810	L	12-11-50	46	19	34	22	+12	+24	0
B	2811	L	12-11-50	57	17	32	28	+4	+29	-3
B	2812	R	12-12-50	74	22	37	40	-3	+34	0
A	2813	L	12-12-50	44	11	26	25	+1	+19	+7
A	2814	R	12-14-50	48	11	26	23	+3	+25	+3
A	2815	L	12-15-50	49	14	29	25	+4	+24	-1
B	2816	R	12-15-50	67	23	38	37	+1	+30	+1
B	2817	L	12-19-50	53	19	34	33	+1	+20	-3
A	2819	R	1-2-51	59	13	28	30	-2	+29	-3
A	2820	L	1-4-51	59	13	28	30	-2	+29	-4
A	2821	R	1-4-51	58	13	28	22	+6	+36	-4
A	2822	R	1-4-51	70	15	30	43	-13	+27	-6
A	2823	R	1-5-51	58	8	23	25	-2	+33	+5
A	2824	L	1-5-51	54	11	26	28	-2	+26	0
B	2825	R	1-8-51	57	24	39	38	+1	+19	-7
A	2826	L	1-8-51	44	15	30	25	+5	+19	+6
A	2827	L	1-9-51	46	7	22	18	+4	+28	-4
A	2828	R	1-11-51	49	10	25	23	+2	+26	+1
A	2829	R	1-15-51	48	12	27	27	+0	+21	-7
B	2830	R	1-16-51	57	23	38	75	-37	-18	+6
A	2831	L	1-19-51	53	8	23	28	-5	+25	-1
A	2833	L	1-23-51	52	14	29	28	+1	+24	-2

CASE	FENESTRATION AND SIDE		DATE	AVERAGE PRE-OP LEVEL FOR 512, 1024, 2048 AC	Bc	ESTIMATE OF POST-OP RESULT	ACTUAL 6 MOS. RESULT	DISCREPANCY BETWEEN ESTIMATE & ACTUAL	GAIN OR LOSS IN db AFTER FEN.	UNOPER. EAR.
B	2835	R	1-22-51	59	17	32	22	+10	+37	+6
B	2836	L	1-25-51	48	21	36	32	+4	+16	+2
A	2837	L	1-25-51	60	7	22	30	-8	+30	0
C	2838	R	1-25-51	83	29	44	95	-51	-12	-18
A	2839	R	1-26-51	58	14	29	37	-8	+21	-3
A	2840	R	1-26-51	56	13	28	35	-7	+21	-8
A	2841	R	1-29-51	57	13	28	35	-7	+22	-2
A	2842	L	1-29-51	47	14	29	25	+4	+22	-8
A	2843	R	1-30-51	47	14	29	18	+11	+29	+2
C	2844	L	1-30-51	74	35	50	62	-12	+12	+8
A	2845	L	2-1-51	53	10	25	28	-3	+25	-1
C	2846	R	2-1-51	85	33	48	63	-15	+22	-3
A	2847	L	2-3-51	68	16	31	25	+6	+43	-1
A	2848	R	2-5-51	59	14	29	37	-8	+22	+5
A	2849	R	2-5-51	36	11	26	20	+6	+16	+12
A	2851	L	2-8-51	63	10	25	23	+2	+40	+6
A	2852	R	2-9-51	44	8	23	20	+3	+24	-6
A	2853	R	2-12-51	55	10	25	30	-5	+25	-1
B	2854	L	2-15-51	73	20	35	28	+7	+45	+5
A	2855	L	2-16-51	45	15	30	30	+0	+15	+1
B	2857	L	2-19-51	62	19	34	38	-4	+24	-3
B	2859	R	2-22-51	56	23	38	25	+13	+31	+0
C	2860	R	2-23-51	89	42	57	52	+5	+37	+7
A	2861	R	2-23-51	58	5	20	28	-8	+30	-1
A	2862	R	2-26-51	57	14	29	22	+7	+35	+1
A	2863	R	2-26-51	49	11	26	28	-2	+21	-1
A	2865	L	2-27-51	46	12	27	22	+5	+24	+3
A	2866	L	2-27-51	64	15	30	40	-10	+24	+3
A	2869	L	3-5-51	49	13	28	35	-7	+14	-2
A	2870	L	3-5-51	47	13	28	27	+1	+20	-1
B	2871	L	3-5-51	53	24	39	38	+1	+15	+4
B	2872	R	3-6-51	63	22	37	32	+5	+31	+5
B	2873	R	3-8-51	73	23	38	37	+1	+36	+5
A	2875	L	3-16-51	36	10	25	23	+2	+13	+6
B	2876	R	3-19-51	82	19	34	50	-16	+32	+4
B	2877	R	3-19-51	56	21	36	33	+3	+23	+2
B	2878	R	3-20-51	68	14	29	42	-13	+26	+0
A	2879	L	3-22-51	49	7	22	30	-8	+19	-1
A	2881	L	3-26-51	67	14	29	25	+4	+42	+5
A	2883	R	3-27-51	65	17	32	33	-1	+32	+2
A	2884	R	3-27-51	49	12	27	28	-1	+21	-2
C	2887	R	3-30-51	65	27	42	45	-3	+20	-4

CASE	FENESTRATION AND SIDE		DATE	AVERAGE PRE-OP LEVEL FOR 512, 1024, 2048 AC	BC	ESTIMATE OF POST-OP RESULT	ACTUAL 6 MOS. RESULT	DISCREPANCY BETWEEN ESTIMATE & ACTUAL	GAIN OR LOSS IN db AFTER FEN.	UNOPER. EAR.
C	2888	L	4-2-51	64	27	42	40	+2	+24	+3
A	2891	L	4-5-51	57	9	24	22	+2	+35	+7
A	2894	R	4-6-51	55	17	32	30	+2	+25	+3
B	2896	R	4-9-51	72	24	39	57	-18	+15	+3
A	2897	R	4-10-51	49	14	29	15	+14	+34	-3
B	2898	R	4-12-51	48	17	32	25	+7	+23	+0
A	2899	R	4-16-51	48	15	30	23	+7	+25	+4
B	2900	R	4-16-51	62	18	33	32	+1	+30	+2
A	2901	R	4-17-51	44	3	18	22	-4	+22	+6
A	2902	R	4-19-51	53	14	29	27	+2	+26	+1
A	2904	R	4-20-51	45	15	30	30	+0	+15	+7
A	2905	R	4-23-51	54	13	28	50	-22	+4	-3
A	2908	R	4-26-51	53	13	28	27	+1	+26	+2
B	2909	R	4-27-51	69	19	34	30	+4	+39	+9
C	2911	R	5-1-51	72	15	30	72	-42	+0	-2
A	2913	R	5-7-51	47	13	28	28	+0	+19	-2
A	2915	L	5-14-51	48	16	31	27	+4	+21	+1
A	2917	R	5-14-51	53	6	21	33	-12	+20	+0
B	2922	L	5-18-51	72	21	36	37	-1	+34	+4
A	2923	L	5-21-51	55	12	27	23	+4	+32	-2
A	2927	R	5-22-51	45	10	25	28	-3	+17	-3
B	2931	R	5-28-51	53	24	39	52	-13	+1	-2
A	2935	R	6-5-51	45	9	24	17	+7	+28	+7
B	2936	L	6-7-51	67	12	27	28	-1	+39	+5
A	2937	R	6-8-51	48	3	18	17	+1	+31	-1
C	2938	L	6-11-51	87	41	56	77	-21	+10	+7
C	2939	R	6-12-51	60	25	40	40	+0	+20	+2
A	2940	R	6-14-51	71	9	24	20	+4	+51	+9
B	2946	R	6-21-51	55	13	28	27	+1	+28	+7
A	2948	R	6-22-51	39	9	24	15	+9	+24	+5
B	2951	R	6-25-51	59	18	33	32	+1	+27	+2
A	2952	L	6-25-51	64	13	28	22	+6	+42	+5
A	2953	R	6-25-51	54	11	26	18	+8	+36	+3
A	2962	R	7-2-51	43	3	18	20	-2	+23	-3
A	2963	R	7-2-51	50	7	22	23	-1	+27	+4
A	2968	R	7-10-51	46	2	17	23	-6	+23	+5
B	2969	L	7-12-51	55	17	32	37	-5	+18	+2
B	2976	L	7-17-51	55	30	45	53	-8	+2	+5
A	2978	L	7-19-51	54	12	27	27	+0	+27	+3

are averaged and corrected for the Carhart notch (see Fig. 2). The predicted result of fenestration is a hearing level 25 db below the corrected bone curve. Since the correction for the notch averages 10 db for the speech frequencies, one may simply predict the result of fenestration in a particular patient as a hearing level 15 db below the average audiometric bone readings for 512, 1,024 and 2,048.

Pt's Name Mrs. R.K. #2369 — Rt. Ear
Surgeon Dr. S. — Left Ear Operated 10-16-50

Date	RIGHT EAR 64	128	256	512	1024	2048	4096	8192	Average for 512, 1024, 2048	Post-op change in db.	LEFT EAR 64	128	256	512	1024	2048	4096	8192	Average for 512, 1024, 2048	Post-op change in db.
5-29-50 Air	40	40	50	45	40	35	30	45			50	50	50	55	50	40	25	52		
10-2-50 Air	40	45	50	50	40	40	30	47			55	65	60	60	60	45	25	53		
10-13-50 Air	40	40	40	50	30	35	20	40			50	50	50	55	45	40	30	50		
5-29-50 Bone	10	20	15	15	15	W/m					10	10	15	20	10	W/m				
10-2-50 Bone	10	20	15	20	20	W/m					5	5	15	20	15	W/m				
10-13-50 Bone	10	15	15	20	20	W/m					0	-5	15	20	15	W/m				
Average Bone											5	3	15	20	13					
Bone Corrected For Otosclerotic Notch											5	-2	5	5	8					
Anticipated Result											30	23	30	30	33				28	
4-16-51	45	45	45	50	35	30	30	43			40	30	30	30	25	30	25	28		
(6 Months Post-operative)																				
10-12-51	50	45	50	50	30	25	45	43			40	30	30	35	20	30	25	28		
(1 Year Post-operative)																				

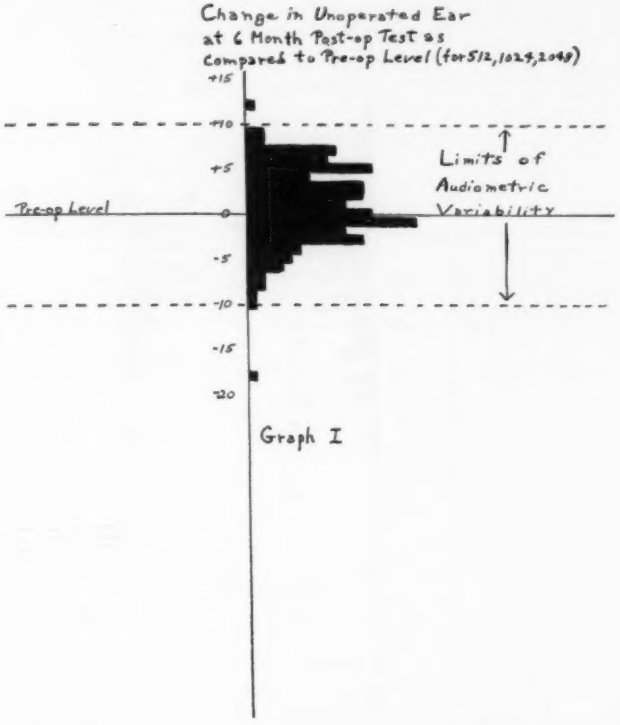
Fig. 2. Calculation of anticipated result of fenestration. The average bone loss for 512, 1,024 and 2,048 in three preoperative audiograms is corrected for the otosclerotic notch. The anticipated result is 25 db below the corrected bone curve. In this patient the actual result six months after operation was the same as the anticipated result.

STATISTICAL STUDY OF THE PREDICTED AND ACTUAL RESULT IN 164 CASES.

The simple formula for predicting the result of fenestration has been applied preoperatively to all cases operated at Northwestern University during the last 15 months.

To test the correctness and validity of the formula, the actual result six months after fenestration when the hearing has become fairly well stabilized was compared with the predicted result. A total of 164 cases was available for study, these cases being consecutive except for a few where we did not have an audiogram at approximately six months after operation.

There were 108 "A" cases of ideal suitability because of bone conducted hearing within the normal range for the speech frequencies of 512, 1,024 and 2,048. There were 43 "B" cases of good but not perfect suitability because of early cochlear involvement with bone conducted hearing below nor-

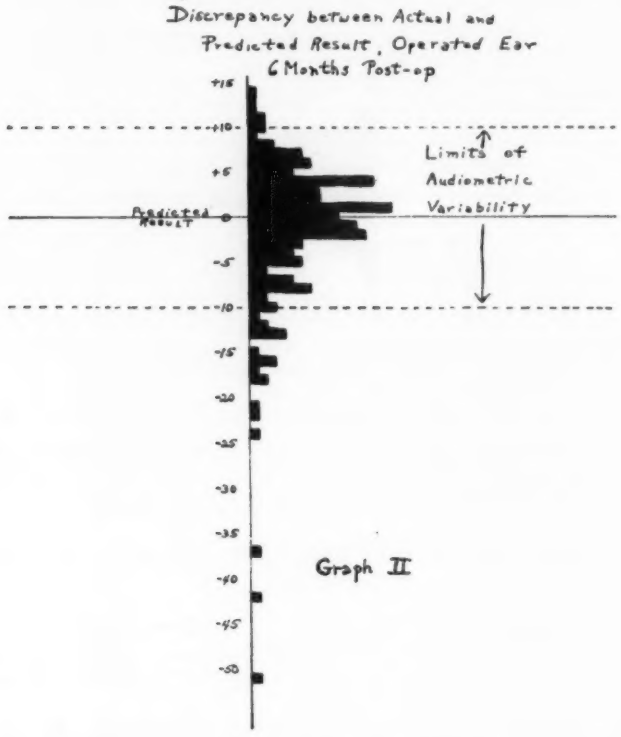


Graph I

mal for 2,048. There were 13 "C" cases of borderline suitability because of moderate cochlear involvement with bone conducted hearing below normal for two or three of the speech frequencies.

The significant audiometric data on each case is summarized, with the classification ("A," "B" or "C") in the first

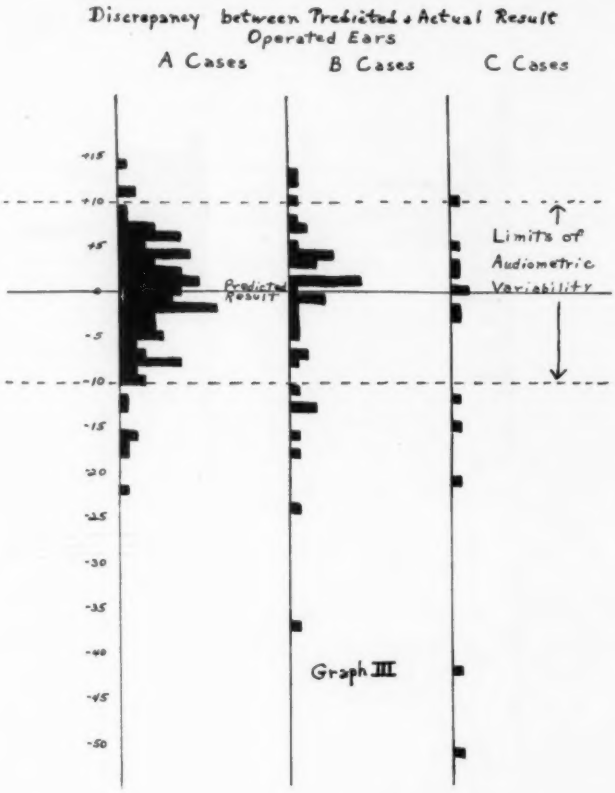
column; the second column is the patient's number in the Northwestern fenestration series; third is the side operated; fourth is the date of operation; fifth is the average preoperative loss for the speech frequencies by air conduction; sixth is the average loss before operation by bone conduction; seventh



Graph II

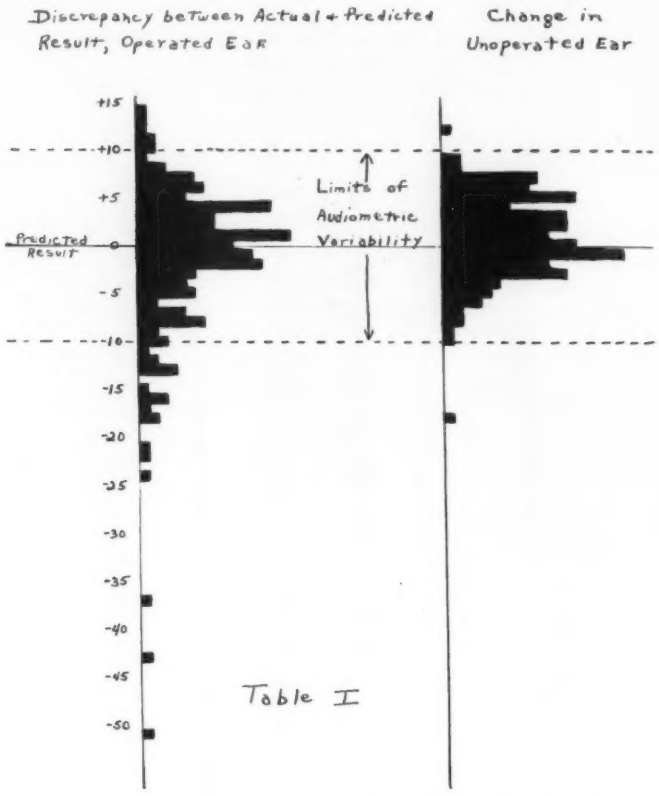
is the predicted result (15 db below the average bone loss); eighth is the actual result at six-month test; ninth is the discrepancy between the predicted and the actual result; tenth is the gain or loss in the operated ear; and the eleventh and last column is the gain or loss in the unoperated ear.

The first graph shows the hearing in the *unoperated* ear six months after operation as compared with the preoperative level. Each square horizontally represents 1 db. Observe how the cases are bunched within several decibels of the preopera-



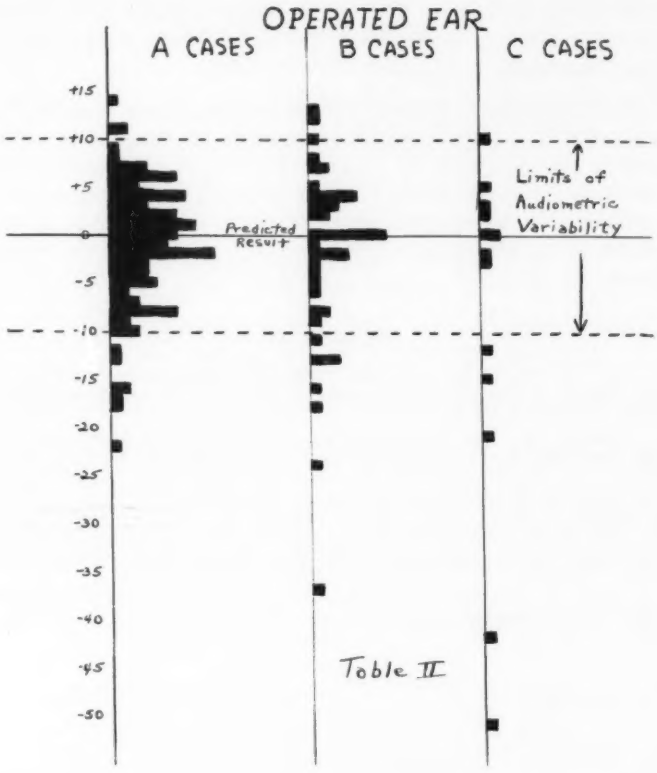
tive hearing ("0"), being distributed fairly equally on both sides. This confirms the observation previously made that *fenestration does not change the hearing in the unoperated ear.* Observe also that with three exceptions the hearing in

the unoperated ear was within 10 db of the preoperative level, suggesting that 10 db may be taken as the average limits of variability in either direction of the hearing level of a patient with otosclerosis.



On the second graph the actual six months result is compared with the predicted result of fenestration. Observe how closely the bunching of results resembles the bunching seen in the graph of the unoperated ear, but with two notable exceptions. First, we see that there is a little wider spread

on either side of the 10 db limit of audiometric variability. This might be due to variations in the depth of the otosclerotic notch of Carhart, not taken into account by the average correction. Thus a shallow notch would be overcorrected, and the predicted result would be too high, and a deep notch would



be undercorrected, so that the predicted result would be too low. Second, we see that there is a sprinkling of cases, totalling 12, where the actual result falls short of the predicted result by more than 15 db. These can be regarded as failures,

if we assume that the formula is correct, even though some of them may show a significant gain in hearing. For example, Patient 2896 gained 15 db of hearing for the speech frequencies but fell 18 db short of the predicted gain.

In the third graph the predicted and actual results are broken down according to preoperative classification. It is interesting to observe that the incidence of failures is significantly greater in the cases of "B" and "C" suitability than in the ideal cases.

From this study it appears that the result of a fenestration operation in a particular patient may be predicted with a fair degree of accuracy by means of a simple formula, with allowance for the variability of repeated audiograms in a patient with otosclerosis and for possible variations in the depth of the otosclerotic notch in the bone conduction audiogram.

REFERENCES.

1. SHAMBAUGH, G. E., JR.: Pure Tone and Controlled Speech Tests in the Election of Cases for Fenestration, International Course in Audiology. *Acta Otolaryngol.* (in press).

2. CARHART R.: The Use of Pure Tone and Speech Audiometry in Otologic Practice. *Trans. Pa. Acad. Opth. and Otolaryngol.*, 4:3:23-40, Oct., 1951.

3. CARHART, R., and HAYES, C.: Clinical Reliability of Bone Conduction Audiometry. THE LARYNGOSCOPE, 59:1084-1101, Oct., 1949.

4. JUERS, A. L.: Observations on Bone Conduction in Fenestration Cases. *Ann. Otol., Rhinol. and Laryngol.*, 57:28, Mar., 1948.

5. DAVIS, H., and WALSH, T.: The Limits of Improvement of Hearing Following the Fenestration Operation. THE LARYNGOSCOPE, 60:273-295, 1950.

CARCINOMA OF THE LARYNX.

A Report of 194 Cases with 149 Laryngectomies.*

JULIUS W. MCCALL, M.D.,
and
WILLIAM R. FISHER, M.D.,
Cleveland, Ohio.

This is a review of the results obtained at St. Luke's Hospital in 194 cases of cancer of the larynx from 1936 through 1950.

TABLE 1.

Total number of cases	194
No treatment or irradiation alone	27
Arbuckle	9
Laryngofissure	14
Laryngectomy	149
Radical neck dissection	18

Of this group, 27 had irradiation alone or refused treatment altogether. There were nine Arbuckle procedures. Fourteen laryngofissures were done, five of which were followed by laryngectomy. There were 149 laryngectomies and 18 radical neck dissections either at the time of or following laryngectomy.

TABLE 2.

SEX.

Total	195	
Male	187	96%
Female	8	4%

*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., Atlanta, Ga., Jan. 13, 1952.

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There was a marked white male predominance, 186 being in this group; seven white females and one each colored, male and female.

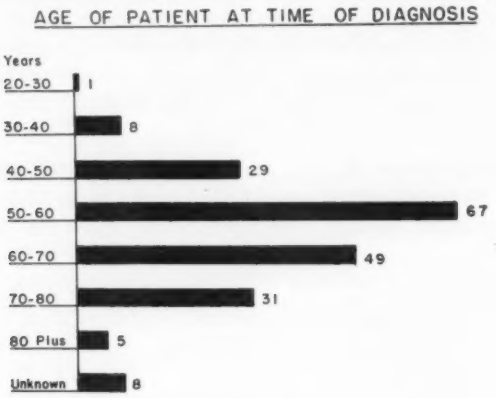

Fig. 1.

The age at time of diagnosis ranged from the third to the ninth decade with a predilection for the 50 to 70 age group. The youngest was a woman of 24, who was six months' pregnant. Following laryngectomy she went on to term, had a normal child and has since had another.

TABLE 3.
HOARSENESS AS A SYMPTOM.

	Int.	Ext.	Total
Alone	33%	24%	57%
With other S-S	7%	22%	29%
Total	40%	46%	86%

Hoarseness was the predominant symptom, occurring as the only symptom in 57 per cent of all cases and in company with others in 29 per cent. Of all patients with a five-year survival without recurrence, hoarseness was the only symptom in 70 per cent (23 out of 33).

TABLE 4.
SYMPTOMS AND THEIR INCIDENCE.

	Int.	Ext.	Total	%
Hoarseness	71	82	153	86
Pain	4	29	33	19
—alone	1	4	—	—
Hemoptysis	—	9	9	5
—alone	—	1	—	—
Dysphagia	2	25	27	15
Cough	6	13	19	11
Dyspnea	5	13	18	10
Palpable mass noted by the patient	—	13	13	7
Weight loss noted by the patient	2	10	12	7

Pain was the next most common, occurring in 19 per cent, dysphagia occurred in 15 per cent. It should be our duty to advise our colleagues, friends, patients and the general public that hoarseness is a serious problem and should be investigated.

TABLE 5.
LARYNGOFISSURES.

Total	11	
Laryngectomy afterwards	3	
Free of disease		
Less than 1 year		2
1-2 years		1
2-3 years		1
4-5 years		2
7 years		1
		7

There were 14 laryngofissures in this series, three of which were done elsewhere and followed here. One of these died of recurrence in less than two years, and two had laryngectomy for recurrence, one year and 12 years later. There is no follow-up on one additional patient. Of the remaining 10 cases done here, one had laryngectomy because of incomplete resection on microscopic inspection, and two required laryngectomy for recurrence two and six years after fissure. The remaining seven patients are free of disease, four months to seven years

after surgery. Two patients in this series received X-radiation and both recurred. Although there were only 14 laryngofissures in this series, the fact that five later had laryngectomy shows they were not selected carefully enough.

TABLE 6.
ARBUCKLE PROCEDURE.

Total	9
Dead of cancer	6
Dead, no evidence of cancer	1
Alive without recurrence	2

The Arbuckle procedure was performed on nine patients by some of the younger men to appraise its worth. In fairness we will state that eight of these cases were far advanced. Five of these patients were dead in less than six months; one due to pneumonia and crusts blocking the tracheal stoma, no evidence of carcinoma being found at autopsy. The other four died of carcinoma. Two additional patients died of cancer in less than a year and one-half. Two patients are living and apparently free of disease three years after treatment. Following the procedure there was one esophagotracheal fistula, one esophageal stenosis, and one chronic, crusting tracheitis.

There were 149 laryngectomies, 86 of which were done five or more years ago. All were done under general endotracheal anesthesia with instillation of 4 per cent cocaine into the trachea and infiltration of the skin and the immediate operative field with novocaine 1 per cent containing 5 to 15 minums of epinephrine per ounce, depending upon the patient's preoperative blood pressure and cardiac status. From 1936 through 1941, avertin was used for basal anesthesia; from 1942 through 1950, pentothal, alone or in combination with nitrous oxide, was used as the anesthetic of choice. A high concentration of oxygen was applied to the endotracheal tube throughout the operation except during the actual work on the tracheal stoma. After 1947, novocaine block of the cervical plexus was performed by the anesthesia department before the start of the operation in nearly all cases.

The 86 patients laryngectomized from 1936 through 1946 are divided into intrinsic and extrinsic. For this report, the intrinsic are defined as involving one or more of the structures between and including the true and the false vocal cords, from the thyroid cartilage anteriorly to the arytenoids posteriorly but not invading either. All others are extrinsic, including spread to any of the cartilages, epiglottis, neck, pharynx, esophagus, or subglottically to the trachea. There were 50 extrinsic and 36 intrinsic. There were a further 19 cases excluded from the analysis because of death during the initial hospital stay (four, all extrinsic), death after discharge not due to the operation or the cancer (two intrinsic, four extrinsic), and the inability to follow the patient for five years (six intrinsic, eight extrinsic).

TABLE 7.

INTRINSIC TREATED BY LARYNGECTOMY.
1936-1946, Inclusive.

Total	28	
Death due to cancer	3	
Five-year survival without recurrence	25	90%

Of the remaining 28 intrinsic, one patient died of recurrence in less than six months, one died of recurrence more than 10 years later, and two died of recurrence but the date is unknown; one lived more than five years without disease but had developed recurrences when last seen. Twenty-three have lived more than five years without disease, and counting the two above who developed recurrences more than five years

TABLE 8.

INTRINSIC TREATED BY LARYNGECTOMY.
1936-1941, Inclusive.

Total	7	
Deaths due to cancer	2	
Ten-year survival		
Without recurrence	5	71%

after surgery, the five-year survival without recurrence for intrinsic carcinoma of the larynx treated by laryngectomy is 90 per cent in this series.

There are nine cases among the intrinsics which were done more than 10 years ago. Two were last seen between five and 10 years after surgery and had no evidence of cancer at that time. Three died of recurrence, one in less than a year, one in his fifteenth year, and the date is unknown for the other. Four of the seven followed more than 10 years are still living and are free of disease; one, 15 years later; one, 13; and two, 10 years later.

TABLE 9.
EXTRINSIC TREATED BY LARYNGECTOMY.
1936-1946, Inclusive.

Total		34	
Deaths due to cancer inless than five years		24	
Alive with recurrence in less than five years		3	
Five-year survival			
Without recurrence		7	21%
Total incompletely resected		17	
Death unrelated		1	
No follow-up		1	
Death due to recurrence			
Less than two years	11		
2-3 years	1		
Date unknown	2		
	—	14	
Alive with recurrence in less than two years		1	
Five-year survival for extrinsic completely resected			37%

For the remaining 34 extrinsic cases with a five-year follow-up the results are much more depressing. Twenty-six patients died of carcinoma, only two of whom survived more than five years. Three patients had metastases when last seen less than four years after laryngectomy. Only five patients were alive without recurrence when last seen more than five years later. Counting the two who developed recurrences after five years, the five-year survival without recurrence in extrinsic carcinoma treated by laryngectomy is only 21 per cent in this series.

In 17 cases carcinoma was left at the time of surgery; either it was visible grossly but could not be removed, or on microscopic examination the tumor was found to extend to the line of resection. Adjusting the values for extrinsic carcinoma to include only those in which all the cancer was apparently removed at surgery, the five-year survival rate for extrinsic carcinoma is still only 37 per cent. There is one 15-year survival among these.

TABLE 10.

CARCINOMA TREATED BY LARYNGECTOMY.
1947-1950, Inclusive.

Total	63
Dead of unrelated causes	2
Dead of carcinoma	12
Living with recurrence	4
Living without recurrence	45

From 1946 through 1950 there were 63 laryngectomies done here. Twelve patients are dead of recurrences, four are living with recurrences and two are dead of unrelated causes. The others were living without obvious cancer when last seen.

TABLE 11.

Cases diagnosed, 1936-1946:	
Intrinsic	41%
Extrinsic	59%
1947-1950:	
Intrinsic	50%
Extrinsic	50%

From 1936 through 1946 there were 41 cases of intrinsic and 59 cases of extrinsic carcinoma diagnosed at this hospital. From 1947 through 1950, there were 42 cases of intrinsic and 42 cases of extrinsic carcinoma. This indicates that physicians and the general public are recognizing the seriousness of hoarseness and are investigating it earlier.

TABLE 12.
RADICAL NECK DISSECTIONS.

Total done on 16 patients		18
Dead of recurrence 2 years later		8
Living with recurrence 2 years later		2
Death in hospital		1
Alive without recurrence		5
Less than 1 year	2	
1-2 years	1	
3-4 years	2	

Eighteen radical neck dissections were done on 16 patients. Ten were done at the time of laryngectomy and eight were done for recurrences up to a year and one-half later. Eight were dead less than two years after the dissection, two were living with recurrences in less than two years, one died in the hospital during the initial stay, and five are living without recurrences; two less than a year, one 27 months, and two are in their fourth year. It is felt that these were done too seldom and too late. Neck dissection should be done for all cases of extrinsic carcinoma at the time of laryngectomy even in the absence of palpable nodes.

The combined five-year survival rate without recurrence for laryngectomized patients in this series is 52 per cent; or, excluding those 17 in whom the tumor was incompletely resected, the rate rises to 73 per cent. Jackson,[1] in 1945, reported a five-year survival of 75 per cent in intrinsic carcinomas treated by fissure or laryngectomy, and a 64 per cent five-year cure rate for all types of treatment, whether intrinsic or extrinsic. In 1948, Schall[2] reported a 64 per cent survival for laryngectomized patients. Clerf,[3] also in 1948, reported a 64 per cent overall survival; and survival without recurrences in five years of 58.7 per cent. Martin[4] has reported 31 per cent five-year cures in intrinsic and 10 per cent in extrinsic. Many of the patients in this series were admittedly poor risks. both as to general conditions of age, heart, hypertension, etc., and as to the possibility of cure of the cancer, but it was felt they should be given the chance of one or two years of extra life they would otherwise not have had.

TABLE 13.
QUALITY OF ESOPHAGEAL VOICE.

With	Preop. Train	Without
67, or 68%	Good	13, or 48%
16, or 16%	Poor	1, or 4%
16, or 16%	None	13, or 48%

Preoperative training in belching, even for a day, is one of the most important factors in the development of an esophageal voice.[5] The ability to be understood over the telephone is taken as the gauge of a good esophageal voice. One which cannot be so understood but which is understandable directly is a poor voice. Of 99 patients receiving some form of preoperative training in belching, 68 per cent developed a good voice as opposed to 48 per cent who received no such training. Sixteen per cent receiving training developed poor voices and the same per cent had no voice, as compared to 4 and 48 per cent for the 27 receiving no training.

TABLE 14.
ADDITIONAL PRIMARY MALIGNANCIES.

Carcinoma of the esophagus	2
Leucemia	1
Malignant follicular lymphoma	1

Additional primary malignancies developed in four patients in addition to the carcinoma of the larynx. One had a malignant lymphoma of the larynx and the cervical nodes at the time of laryngectomy, one developed leucemia, and two developed primary carcinoma of the esophagus.

One of the latter was inoperable when diagnosed seven years after laryngectomy; the other had an esophagectomy and still maintained an understandable voice. An additional patient developed a solitary pulmonary metastasis, underwent pneumonectomy four years after laryngectomy, and is again

free of disease one year later. Two laryngectomized patients drowned when their fishing boats overturned; water sports are an added hazard in these cases and the patient should be so informed.

SUMMARY.

1. One hundred ninety-four cases of cancer of the larynx are reported.

2. Hoarseness was the only symptom in 70 per cent of the five-year survivals.

3. There were 14 laryngofissures. Five had laryngectomy later. One died of recurrence. Seven are living without recurrence at the present time.

4. Of nine Arbuckle procedures, only two patients are living and free of disease two to three years later.

5. One hundred forty-nine laryngectomies were done under general anesthesia with no immediate postoperative deaths. There were eight deaths during the initial hospital stay.

6. Of the intrinsic carcinomas treated by laryngectomy and followed for five or more years, 90 per cent survived more than five years without evidence of disease. Of seven patients with a 10-year follow-up, four are living without cancer.

7. In extrinsic carcinomas, the five-year survival rate was only 21 per cent.

8. The overall five-year survival rate for laryngectomized patients was 52 per cent.

9. Carcinoma of the larynx is being seen earlier at this hospital.

We thank Dr. F. W. Alexander, Dr. L. C. Dickey, Dr. F. S. Gardiner, Dr. A. J. Lamb, Dr. P. M. Moore and Dr. H. C. Rosenberger for their help in obtaining follow-up data on their patients included in this series, and Mr. Fred Beale for his help in preparing the tables used in this presentation.

REFERENCES.

1. Jackson, Chevalier L., and Norris, Charles M.: Surgical Treatment of Cancer of the Larynx. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, p. 45, 1945.

2. Schall, Leroy A., and Ayash, John J.: Cancer of the Larynx. *Ann. Otol., Rhinol. and Laryngol.*, 57:377, 1948.

3. Clerf, Louis H.; Putney, F. Johnson, and O'Keefe, John J.: Carcinoma of the Larynx. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, p. 482, 1948.

4. Martin, Hayes: Cancer of the Head and Neck. *Jour. A. M. A.*, 137: 1306, Aug. 7, 1948.

5. McCall, Julius W.: Preliminary Voice Training for Laryngectomy. *Arch. Otolaryngol.*, 38:10-16, July, 1943.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The joint meeting of the North Carolina Eye, Ear, Nose and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held in Winston-Salem, N. C., Sept. 15-17. Headquarters: Robert E. Lee Hotel in Winston-Salem, with the following guest ophthalmologists: Dr. P. Robb McDonald, Philadelphia, Pa.; Dr. Irving H. Leopold, Philadelphia, Pa.; Dr. Harold W. Brown, New York, N. Y., and Dr. Frank W. Newell, Chicago, Ill.

The guest otolaryngologists include Dr. Theodore E. Walsh, St. Louis, Mo.; Dr. Frank Tourschik, Bridgeport, Conn., and a third otolaryngologist to be announced later.

For further information please communicate with Dr. G. B. Ferguson, Secretary of the North Carolina Eye, Ear, Nose and Throat Society, c/o The McPherson Hospital, 1110 West Main Street, Durham, N. C., or with Dr. Roderick Macdonald, Secretary of the South Carolina Society of Ophthalmology and Otolaryngology, 330 East Main Street. Rock Hill, S. C.

AN AFFERENT REPRESENTATION OF THE LARYNX IN THE CEREBELLUM.

ROBERT L. LAM, M.D.,* and JOSEPH H. OGURA, M.D.,†
St. Louis, Mo.

INTRODUCTION.

It is obvious to those concerned with laryngeal disease that important central connections must exist in relation to the afferent nerve supply of the larynx. Laryngeal functions in both health and disease, particularly cough and reflex closure of the vocal cords, autonomic (neuromuscular, neurovascular and neurosecretory) reflex activity, appreciation of specific sensations, maintenance of cord tonus, and the delicate control of phonation—these complex functions presuppose a diverse system of neuronal centers and pathways in the cerebrospinal axis. In all probability, reflex arcs predominate at the brainstem and cerebellar levels, while conscious apperception of various modalities rest in the thalamus and cerebral cortex; yet, neuroanatomical or electrophysiological evidence for these tracts is practically unknown and, therefore, the present investigation was carried out to demonstrate the rôle that hindbrain structures may play in the synaptic mediation of impulses derived from laryngeal receptors.

The superior laryngeal nerve, well accepted as the principal sensory supply of the larynx, was selected for afferent stimulation. To study medullary and cerebellary projection areas, specific electrical responses were recorded after the proximal segment of the cut superior laryngeal nerve of the cat was stimulated by single shocks. Consistent potentials were found

*Department of Neuropsychiatry, Washington University School of Medicine, St. Louis, Mo.

†Department of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.

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in two discrete areas of the cerebellar cortex: crus I of the ansiform lobule bilaterally, and the third and fourth folia of the paramedian lobule bilaterally (see Fig. 1).

METHOD.

Twelve cats weighing 2.0 to 2.5 kg. were given 30 mgm./kg. body weight of veterinary nembutal intraperiotoneally. A stable level of anesthesia of moderate depth was maintained by the addition of endotracheal ether through a low tracheotomy. Extension of the longitudinal incision from the mandible to the supraclavicular notch afforded excellent exposure of the cervical vagus, nodose ganglion, and superior laryngeal nerve. After immobilization in a head holder, each animal was rotated 75 degrees along its longitudinal axis in a specially devised rack. In this position both the cerebellum and the superior laryngeal nerve could be approached with ease.

The superior laryngeal nerve was dissected free throughout its entire course from the nodose ganglion to the thyrohyoid membrane. The external or motor branch to the cricothyroid muscle was identified and separated from the larger sensory branch before the latter was severed. The proximal segment of that nerve was then placed upon No. 24 nichrome wire electrodes, completely insulated except for actual stimulating surfaces in contact with the nerve. The entire nerve preparation was carefully suspended in air away from other body tissues and protected from drying by mineral oil.

After exposure of the dorsal surface of the cerebellum, a specially-devised microscope mechanical stage mounted upon the headholder permitted exploration of the brain-stem and cerebellum in all principal planes. Silver recording electrodes were insulated except for a 100 μ tip. A critical exploring electrode was led against a periosteal electrode situated remotely to the cerebellum. Spontaneous and cerebellar response potentials were recorded on a cathode ray oscilloscope. Single shocks of varying duration and intensity were delivered to the superior laryngeal nerve through an isolation transformer. The thresholds necessary to activate the differ-

ent types of fibres in the nerve were determined and then compared with those sufficient to evoke the specific cerebellar or brain-stem response. Arbitrarily, threshold stimuli will be referred to by the unit 1, and supraliminal strengths as multiples of 1.

All exposed areas of the dorsal cerebellum were explored, the critical electrode tip just touching the pial surface of the exposed cortex. The vermis, lobulus simplex, crus I and II of lobulus ansiformis, and lobulus paramedianus were accessible to examination. The ventral surfaces were not studied. The medulla was similarly explored in six experiments, the results of which will be reported after more thorough investigation.

RESULTS.

In these experiments, the superior laryngeal nerve of the cat demonstrated two potential components: *1.* a large initial spike potential with conduction rate of 25-30 M/sec., derived from 6-10 μ (cross-sectional diameter) fibres showing a relatively low threshold, designated as 1; *2.* a considerably smaller wave with rate of about 3 M/sec. related to thinly myelinated fibres of 2-4 μ size propagated by stimulus intensity 75. Osmic acid impregnation revealed that the larger fibres accounted for three-fourths of the cross-sectional area of the superior laryngeal nerve.

Corresponding to the activation of the larger myelinated, most rapidly conducting fibres of the superior laryngeal nerve, responses were recorded from remarkably discrete areas of lobulus ansiformis and paramedianus bilaterally. All potential activity recorded from the pial surface of the cerebellum evoked by superior laryngeal nerve stimulation resulted from a relatively brief, low intensity single shock representing the threshold for activation of the first component of the nerve-conducted potentials. No responses could be found correlating with the higher intensities necessary to elicit the second component. Varying durations and strengths of stimuli produced no change in the form of the cerebellar response.

The cerebellar areas from which evoked potentials were recorded were exceedingly small (see Fig. 1). In crus I the locus of responses measured usually 1.0 to 1.5 mm. in diameter; in paramedianus, 0.5 to 0.75 mm. The crus I projection area was consistently in the inferomedial angle anterior to the first folium of paramedianus. Responses of the paramedian

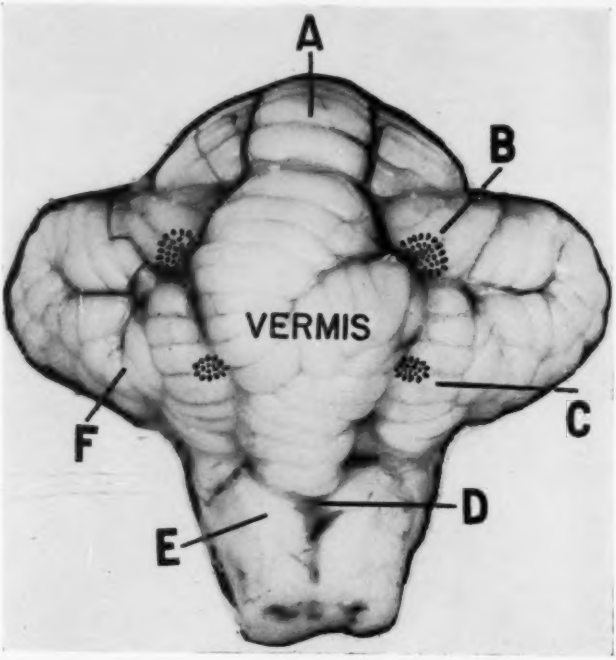

Fig. 1. Photograph of dorsal surface of cat cerebellum. The four stippled areas represent the loci of evoked potentials from afferent stimulation of the superior laryngeal nerve. (A) Anterior lobe. (B) Crus I of lobulus ansiformis. (C) Lobulus paramedianus. (D) IV ventricle. (E) Medulla. (F) Crus II of lobulus ansiformis.

lobule were sharply limited to the medial one-third and the adjacent lips of folia III and IV. Evoked potentials were not noted to arise from any other areas of the dorsal surface of the cerebellum.

The cerebellar responses were most frequently single monophasic positive spikes when the critical electrode was led against the periosteal electrode (see Fig. 2A). This was most constant in the center of the locus. Duplication of this potential (see Fig. 2B) and occasional showers of small multiple spikes superimposed upon the basic response occurred in the periphery of the area activated (see Fig. 2C, D, E).

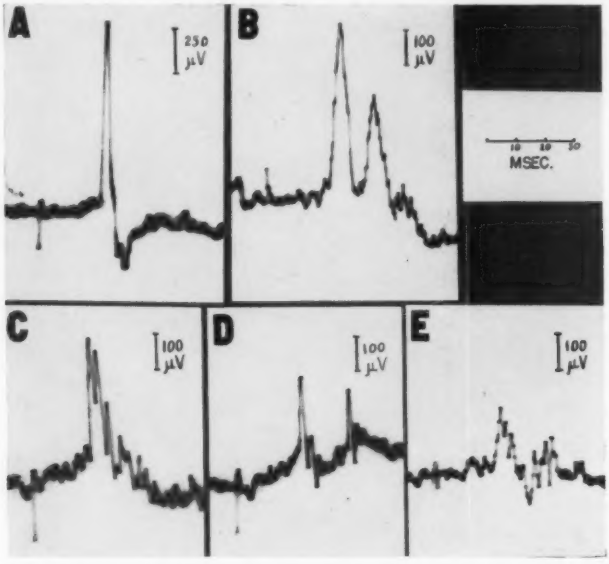

Fig. 2. Photograph showing the various types of evoked cerebellar potentials from superior laryngeal nerve stimulation. These responses were recorded from the posteromedial portion of the right crus I on supraliminal excitation of the ipselateral superior laryngeal nerve in cat No. 1-17-52. (A) Single positive 1 mv. response with a negative 250 μv component, suggestive of diphasicity. The more common evoked potentials were entirely monophasic. (B) Duplicated positive 450 μv and 250 μv responses. (C), (D) and (E) Multiple spikes; there is superimposition upon a larger and more basic response in (C) and (E). The multiple spike potentials were of lower amplitude than the single and double responses, and were recorded in the periphery of the projection area on crus I. The latent period for above responses was 20.0 msec.

Latent periods for the cerebellar response varied from experiment to experiment, due to variation in size of the animal, in temperature and moisture control; however, the latency recorded from stimulation of the superior laryngeal

nerve to the beginning of the evoked cerebellar potential ranged from 20 to 30 msec. A difference of 0.75 to 1.0 msec.

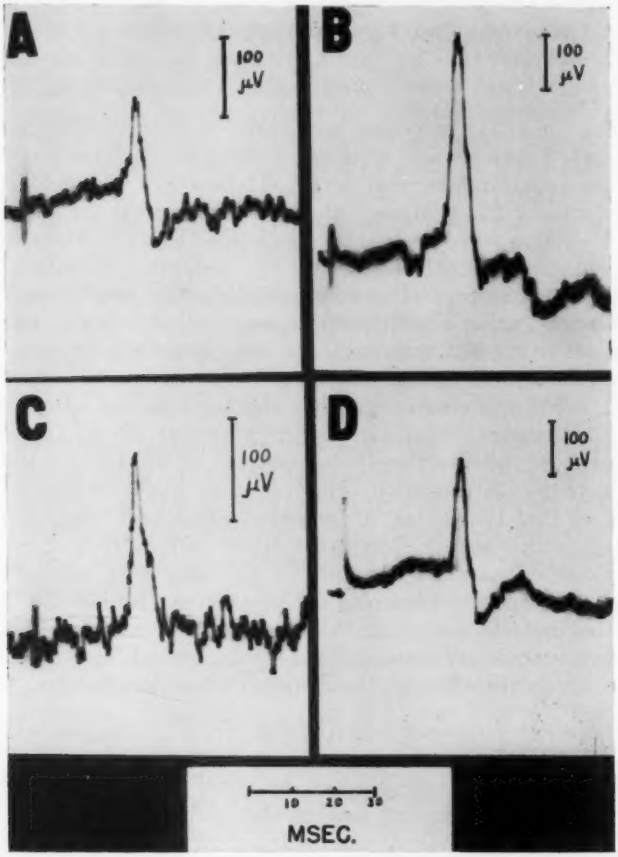

Fig. 3. Photograph illustrating evoked responses from crus I and paramedianus bilaterally in the cerebellum of cat No. 12-12-51. The left superior laryngeal nerve was stimulated. (A) Diphasic positive-negative response from left crus I with latency of 25.7 msec. Stimulus intensity 40. (B) Monophasic positive response from right crus I with latency of 26.5 msec. Stimulus intensity 75. (C) Monophasic positive response from left paramedianus with latency of 23.2 msec. Stimulus intensity 30. (D) Diphasic positive-negative response from right paramedianus with latency of 24.1 msec. This experiment demonstrated evoked potentials from all previously observed laryngeal projection areas in the cerebellum from afferent stimulation of one superior laryngeal nerve.

was noted in the latent periods of contralateral and ipsilateral responses, the latter showing shorter time intervals. Paramedian lobule potentials occurred about 2.0 msec. earlier than crus I responses (see Fig. 3A, B, C, D).

DISCUSSION.

The cerebellar responses correspond to threshold excitation of those moderate size myelinated fibres which form the bulk of the superior laryngeal nerve. These fibres had a conduction rate of 25-30 M/sec., and a cross-sectional diameter of 6-10 μ. The wave component assignable to these fibres may be regarded as included in the "A" potential described for the cat vagus nerve (Heinbecker,[1] Heinbecker and O'Leary[2]). Ordinarily, axones with the size seen in the superior laryngeal nerve are characterized by a more rapid conduction rate of 30-50 M/sec. In the animal preparations used in this report, the slower conduction rates can be explained by cooling, as maintenance of constant temperatures of 37° C. for each experiment was relatively impractical. Such fibres usually mediate tactile sensation. On the other hand, it cannot be ignored that the above "A" potential component may be consistent with visceral afferent functions, because fibre size and myelination may not correlate in all cases with conduction rates and specific physiological properties. Heinbecker has pointed out the fact that "visceral nerve trunks practically always contain myelinated fibres of the second 'fast' somatic type which are afferent, the so-called 'visceral afferents'."

The sensory representation of the larynx in the cerebellum is not altogether surprising in view of the classical studies of Snider[3] showing extensive but delimited areas of the cerebellar cortex of the cat and monkey which are devoted to afferent functions. Dow[4,5] and Adrian[6] have likewise revealed sensory projections to the cerebellum by peripheral nerve stimulation. The loci of specific potential activity in crus I and paramedianus determined by excitation of the superior laryngeal nerve correlate well with the anatomical diagrams drawn by Snider to illustrate sensory projections to the cerebellar cortex. Derived from the fourth and fifth arches as an

evagination of the foregut in embryological development, the larynx should occupy theoretically a position between the face and upper extremity in terms of any sensory scheme projected to the central nervous system. Snider found two separate areas in the cat cerebellum showing electrical responses to tactile stimulation of the face, forelimb and hindlimb: *1.* anterior lobe (posterior portion) overlapping crus I and lobulus simplex; *2.* lobulus paramedianus overlapping crus II. The body schemes were reversed in these two regions: in *1.* the face was most posterior, forelimb intermediate, and hindlimb most anterior. The opposite was true for *2.* in terms of anteroposterior orientation.

The experimental demonstration of laryngeal projections to crus I corresponds closely to Snider's face area; however, its projection to the medial one-third of the adjacent surfaces of the third and fourth folia of paramedian lobule is more in the forelimb area described by Snider.

The shorter latent periods recorded for paramedianus and ipsilateral responses evoked by superior laryngeal nerve excitation suggest the factors of relatively decreased central transmission distance or less synaptic delay. More exact explanation of this phenomenon depends upon more detailed investigation.

Measurement of the distance between the point of excitation on the superior laryngeal nerve (at the proximal or cathodal stimulating electrode) and the site of entrance of vagal rootlets revealed values of 25-31 mm.; thus, since the stimulus intensity of 1 activates the nerve component with a conduction rate of 25-30 M/sec. and also the cerebellar potential at the same time, it can be readily seen that only a small fraction of the total latent period can be related to impulse transmission along the superior laryngeal nerve, nodose ganglion, and proximal vagal radicles. From these observations a complex system with several relays between laryngeal nerve terminals and cerebellum may be inferred.

In some experiments consistent responses were recorded from the medulla, probably indicative of the first synaptic

station in afferent laryngeal functions. Further work will be done to clarify what part these responses may play in central connections of laryngeal afferents.

The exact significance of afferent representation of the larynx in the cerebellum at this time is not clear. These experiments add to the speculation and re-examination of facts concerning cerebellar physiology precipitated in a large part by Snider and his co-workers. The cerebellum probably participates on a subconscious level in the integration and coordination of the fundamental reflex arcs of the larynx. The inflow of afferent discharges to the cerebellum may be highly important in the preservation of tonus in the vocal cords; constant stimulation of nerve endings in the epithelium of the cords in the normal physiological state may result reflexly in producing tension. Irritation of touch and pain fibres in the laryngeal mucosa may produce reflex closure of the vocal cords, cough, or autonomic phenomena, controlled in part by the cerebellum in conjunction with lower brain-stem centers. Smoothly coordinated phonation must infer intact cerebellar pathways, in which sensory projections must represent an integral part; the ataxic, scanning, and explosive speech of the multiple sclerotic is an outstanding example of the results of cerebellar disease reflected in phonatory dysfunction. Afferent cerebellar connections may explain the syndrome of laryngeal vertigo, the pathophysiology of which is obscure.

After more study and evaluation the cerebellum or "Kleinhirn" may actually be a structure less secondary to the cerebrum or "Grosshirn" than previously considered and particularly important in widespread integrative functions embracing all afferent discharges in the organisms.

CONCLUSION.

1. Bilateral afferent laryngeal projections exist in the paramedian lobule and crus I of the ansiform lobule in the cat cerebellum.

2. The cerebellum probably plays a most important and hitherto unemphasized rôle as a coordinator in laryngeal functions.

We acknowledge the invaluable suggestions of George H. Bishop, Ph.D., James L. O'Leary, M.D., and Theo. E. Walsh, M.D., in the preparation of this work.

BIBLIOGRAPHY.

1. Heinbecker, P.: The Potential Analysis of the Turtle and Cat Sympathetic and Vagus Nerve Trunks. *Amer. Jour. Physiol.*, 93:284, 1930.

2. Heinbecker, P., and O'Leary, J. L.: The Mammalian Vagus Nerve—A Functional and Histological Study. *Amer. Jour. Physiol.*, 106:623, 1933.

3. Snider, R. S.: Recent Contributions to the Anatomy and Physiology of the Cerebellum. *Arch. Neurol. and Psychiat.*, 64:196, 1950.

4. Dow, R. S.: Cerebellar Action Potentials in Response to Stimulation of Various Afferent Connections. *Jour. Neurophysiol.*, 2:543, 1939.

5. Dow, R. S., and Anderson, R.: Cerebellar Action Potentials in Response to Stimulation of Proprioceptors and Exteroceptors in the Rat. *Jour. Neurophysiol.*, 5:363, 1942.

6. Adrian, E. D.: Afferent Areas in the Cerebellum Connected with the Limbs. *Brain*, p. 289, 1943.

FIBROUS DYSPLASIA.*

HOWARD MCCART, M.D.,
Toronto, Canada.

Many articles have been written on this changeable disease under other titles, such as osteodystrophia fibrosa, juvenile Paget's disease, leontiasis ossea, osteitis fibrosa, von Recklinghausen's disease of bone, fibrocystic disease and osteitis fibrosa cystica.

The main features of this disease are varied and are not always confined to the skeleton. There appear to be three disconnected features: *1.* skeletal lesions affecting almost every bone in the body or affecting one bone or a few bones, *2.* pigmented patches in the skin, *3.* endocrine disturbances manifested by sexual and somatic precocity, especially in the female. The essential clinical features of this disease, however, are skeletal and may or may not be accompanied by either or both of the other two.

Fibrous dysplasia is one in a group of diseases in which softening, bending and fracture of the bones may occur. In general, this group can be separated into two main divisions. In one, the lesions are generalized in the bones with accentuations here and there which comprise the deficiency and endocrine diseases, rickets, osteomalacia, osteoporosis and hyperparathyroidism; and in this division, specific causes and means of prevention and cure are known. In the other division, the lesions are more or less disseminated in the skeleton or even localized in a single bone comprising Paget's disease, dyschondroplasia, osteogenesis imperfecta and fibrous dysplasia, for none of which is there a known cause or a specific means of cure.

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., New York, N. Y., Jan. 11, 1952.

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In 1885, the pathologic similarity between rickets and osteomalacia was pointed out, but not for many years was the common etiologic factor discovered. In 1877, Sir James Paget thoroughly described the clinical and pathological features of the disease bearing his name (osteitis deformans). The two hereditary diseases, dyschondroplasia and osteogenesis imperfecta, have become well establishd entities. Von Recklinghausen,[1] in 1891, published a report on 16 cases of bone diseases, six cases of which he describd as fibrous osteitis, two of which can be accepted as fibrous dysplasia. He included along with five cases of osteomalacia and rickets a case with widespread skeletal malacia, which is generally accepted as one of hyperparathyroidism, a parathyroid tumor having been subsequently identified from his material from this case. Morton [2] in the United States in 1922, reviewed the literature up to date of that year, 63 cases in all, and recognized two main groups, one of which was fibrous dysplasia and the other, hyperparathyroidism, but dismissed the findings of parathyroid tumor at autopsy as incidental.

Elmslie's[3] reference to a nevus on the back of the neck in one of his cases was the first time that mention was made of extra skeletal features of the disease, and Weil, according to Duff, Murray and Pritchard[10] reported a case of a nine-year-old girl who had pigmented patches on the skin and whose sexual and skeletal development was that of a girl of 15.

About 1925, parathormone was discovered, and soon experimental evidence proved that hyperparathyroidism caused osteitis fibrosa cystica, and at this time a parathyroid tumor was removed with startling results. Then von Recklinghausen's disease and hyperparathyroidism became synonymous, but it was soon noticed that in many of these cases of osteitis fibrosa cystica of von Recklinghausen that neither parathyroid tumor or hyperparathyroidism could be demonstrated.

In 1929, Winter[4] reported a case of generalized osteitis fibrosa cystica in the absence of parathyroid tumor. Hunter and Turnbull[5] were the first to stress the distinction between generalized osteitis fibrosa of hyperparathyroidism and the

osteitis fibrosa in multiple foci, particularly emphasizing that in the focal disease, even though many bones were affected, the condition is totally different from the generalized disease and that exploration of the neck for parathyroid tumors is unjustifiable.

Jaffe,[6] in 1933, reported that he had a residue of cases of osteitis fibrosa cystica without parathyroid tumors, of unidentified entity, and suspected that they had a congenital basis.

In spite of Hunter and Turnbull's reference, in 1931, to the distinctiveness of the entity and how it differed from hyperparathyroidism, confusion between the two diseases prevailed and it was not until 1937, when Albright[7] and collaborators brought order out of the chaos, reporting on seven cases with severe skeletal involvement and skin pigmentation. Of these, four females and one of the boys were considered precocious sexually. All of the three males had previously been explored surgically for parathyroid tumors without any having been found. They carried out thorough investigations, including metabolic and endocrinologic studies, and emphasized the focal nature of the lesions.

Lichenstein,[8] in 1938, reported on eight cases in which there was no evidence of extra skeletal features but stressed the distinctive nature of the disease and its multiple focal distribution in the skeleton.

In 1942, Lichenstein and Jaffe[9] were able to collect 75 cases from the literature and to describe 15 more of their own under the title, "Fibrous Dysplasia of Bone," inferring that the skeletal lesions are not necessarily polyostatic.

Duff, Murray and Pritchard,[10] in reviewing the 256 cases reported in articles published between 1929 and 1949, found that the sexes are equally affected, leaving out Schlumberger's[11] 67 monostotic cases which were mostly in men from the armed forces. There were 83 males and 82 females where the sex was stated. In 148 cases ranging from four months to 70 years of age, the disease could be traced to infancy in seven cases, to childhood in 131, and to adolescence in 10. In

many of the other cases, the appearance of the disease was of long duration. That skeletal lesions can exist unnoticed even in widely disseminated form is shown by the not infrequent accidental discovery of the disease on radiological examination.

The first sign of the disease may be precocious menstruation but more often it is related to the skeleton in the form of a limp, a pain, or bending of a bone, particularly the upper end of the femur, a deformity of the face or skull; if the skull, it may be failing vision that first sends the patient to the physician. As a rule, the patients are reported as having a normal infancy with the exception of premature menstruation. Pigmentation of the skin was not reported as an attracting sign in 256 cases reviewed.

There is considerable variation in the progress of the skeletal lesions; in the more severe forms, deformities may become disabling fairly early in childhood, usually reaching the maximum before maturity. Progressive appearance of the foci in the skeleton is well authenticated insofar as they are demonstrable by symptoms and radiography. Whether the foci are all established in marrow at the same time is not known, but individual foci extend and often coalesce. Continuity of activity may extend into adult life or manifestation of activity may occur only in adult life; reactivation after long periods of quiescence is not uncommon.

There are many variations in the bone lesions, but they are all within the potentiality of the skeletoblastoma to produce fibrous connective tissue, cartilage, bone, and synovial cavities and to shift back and forth between the productive and lytic phases of the metamorphosis of the bones and joints. The process of fibrous dysplasia commences as a fibrous tissue proliferation within the medulla, replacing marrow and absorbing and replacing the cancellous and cortical bone. Metaplastic bone is laid down in this newly formed fibrous tissue and occasionally islands of cartilage are developed and cysts may be formed.

The microscopical appearances are described as newly formed fibrous tissue varying from place to place in its composition. It may consist of small spindle and stellate cells and a fine fibrillar stroma in a loose or whorled arrangement, or it may appear in the form of intertwining bundles of larger spindle cells with plump vesicular nuclei and a varying amount of collagenous stroma. On the other hand, it may be poor in cells and highly collagenous, often retaining a whorled or intertwining pattern. Some areas may appear edematous and contain numerous small cystic spaces. The vascularity varies markedly, the vessels being thin walled and of small caliber. Dilatations of vessels may be seen with extravasation of blood, clumps of multinucleated giant cells and deposits of hemosiderin. Clumps of giant cells may be seen, some with few or many nuclei, and should be looked upon as phagocytes. Osteoclasts may be found about islands of cartilage and around fragments of bone in well vascularized areas, this being a manifestation of the lytic phase of bone building.

Areas of fibrous tissue may escape ossification or bone may be laid down metaplastic in nature, appearing as shaggy small masses or irregular trabeculae, or it may appear in a more closely anastomosing trabecular arrangement, going on to form a dense, uniform, or irregular, fine or coarse honeycomb structure. Osteoblasts may appear along the margins of the metaplastic bone trabecula, leading some writers to the opinion that the process is one of osteoblastic formation, but lamellar bone is not a prominent feature. Most likely the osteoblasts play a part of smoothing off the irregular margins of the metaplastic bone trabeculae.

It appears that there is an erosion of the normal preformed bone by the path of the advancing connective tissue without the presence of osteoclasts, so that the line of demarcation between normal preformed bone and pathological connective tissue is quite sharp. The cortex becomes reduced to a thin layer and may ultimately disappear. The fibrous tissue continues to proliferate, there being no bony reaction on the part of the periosteum except in the advent of fracture.

The skeletal lesions may be a single lesion within a bone or a widely disseminated state with nearly every bone in the body involved. In Duff and collaborators' review, the lesions were monostotic in 28 cases. To date, Schlumberger's 67 cases of monostotic lesions is by far the largest group published. In this group, the ribs were involved in 29 cases, the femur in nine; tibia, eight; maxilla, seven; calvarium, five; mandible, two; humerous, two; ulna, two; vertebra, one; pelvis, one; and the fibula, one.

A flat bone may increase six to eightfold or more in thickness. The thickened and hyperostotic vault of the skull, chiefly as a result of replacement of the outer table, simulates that of Paget's disease. The occipital and frontal bulges and grossly thickened, dense, hyperostotic bone are all very characteristic of the face or skull; thus failing vision may be the patient's first symptom. On the other hand, precocious menstruation may be the first sign. A few may show pigmentation of the skin; if so, it appears in infancy, occurring on the back, buttocks, thighs, shoulders, chest, neck, face, or buccal mucosa scalp. It may occur as small or large patchy areas, singly or in multiple areas of brownish cafe-au-lait or yellowish areas. It occurrence increases with the severity of the disease, but is usually absent in the case of limited bone involvement. Duff and collaborators mention it as occurring in 79 cases. Jaffe, in his review of 90 cases, mentions it in 32 cases, while in Schlumberger's 67 monostotic cases, it was absent, though Bennett has observed it in monostotic lesions.

A very important diagnostic feature of the disease is its focal nature in the skeleton. Uninvolved bones or parts of bones between the foci of disease retain their normal structure, which is in striking contrast to hyperparathyroidism in its effect on the whole skeleton by the process of the absorption of bone. An affected bone may retain its normal bony structure and show only a focal replacement of the marrow by a solid fibrous mass containing more or less bone, or the cortex becomes reduced to a thin layer and may ultimately disappear — the fibrous tissue continues to proliferate, there being no bony reaction on the part of the periosteum except

in the advent of fracture; thus the bone may or may not be expanded. The gross appearance of the bone may be pale, pink or cyanotic and may be gritty to cut as a result of new bone formation within, or areas of marble-like bone may be encountered, being so hard as to turn the edge of an enterotome. Areas of hemorrhage, cartilage, or cysts may be recognized.

Lesions of the skull usually assume a hyperostotic state, usually the base becomes greatly thickened and dense and bulges upward into the cranial cavity. The vault thickens chiefly by replacement of the outer table. The process may be diffuse with the head enlarged symmetrically or it may be focal, causing marked asymmetry. Large occipital and frontal bulges are not uncommon. Extension forward leads to facial deformities and obliterates the sinuses and nasal passages, separating the eyes and displacing them in a horizontal or vertical plane or causing proptosis. Encroachment of the foramina in the base of the skull may cause optic atrophy and diminution in vision and hearing.

In the X-ray, the area of fibrous dysplasia is radiolucent, sometimes transversed by delicate trabeculae of bone, varying according to pattern and distribution of the new bone and its calcification. One may find alternating areas of translucency and increased opacity. It is usually central in position and produces thinning and expansion of the cortex. As cortical absorption progresses, the appearance will be that of a bone covered by a thin cortex and when complete absorption occurs, the core of the fibrous dysplasia will be covered by periosteum with no distinction between cortex and medulla.

Blood chemistry findings are essentially negative; the calcium and phosphorous levels in the blood serum are normal, and a negative calcium balance does not occur in fibrous dysplasia.

In females, precocious menstruation is a common accompaniment of the skeletal lesions and is usually associated with pigmentation of the skin.

In males, sexual precocity is very much less common and was recorded in only five cases against 32 cases in the females in the review by Duff and collaborators.[10]

The majority of the cases were normal mentally, though five patients were described as mentally retarded in Duff's[10] review.

In the past few years we have observed five cases, one or two of which show interesting features.

REPORT OF CASES.

Case 1: N. S., a 19-year-old white male, admitted to hospital January, 1949, complaining of a lump in the right side of the lower jaw for the past two and one-half months and, for the past month, a lump in the left lower jaw. They have gradually increased in size; now are about 6 cm. in diameter and are not tender. Examination shows a firm swelling on external surface of right mandible opposite two premolar teeth, non-tender, measuring about 6 cm. in diameter and not involving the skin. The one in the left mandible is somewhat smaller and situated in about the same area.

X-ray report (see Fig. 1) shows two large radiolucent areas in the ramus of the mandible, the right being larger than the left. On the left side, the radiolucent area involves the ramus of the mandible below the apex of the first molar tooth and seems to extend upward between the second bicuspid and first molar. On the right side, the radiolucent area is a little farther anteriorly, extending from the first bicuspid forward to lateral incisor. The cortex of the mandible is almost paper thin with no evidence of any reaction. It would appear that there are radicular cysts. The fact that the teeth are fully developed and seem to be outside the cyst excludes their being dentigerous cysts.

Operation January, 1949: Lower right cuspid, first and second molars removed, an incision was made along the length of the alveolar crest, revealing thin cortical bone in which was a fibrous mass which could be readily enucleated. The mass was not removed from the left jaw, awaiting biopsy reports on the tissues from the right jaw.

Pathological report (see Fig. 2) shows a dense collection of cells arranged in whorls and streaks for the most part, with abundant cytoplasm and fusiform nuclei. These are probably of fibroblastic origin. Interspersed are collections of cells with more circular and pale staining nuclei, but these are in the minority. There are a few cystic spaces present filled with cellular debris; here and there are small homogenous densities taking a polychromatic stain which resembles bone. No giant cells seen. Diagnosis: fibrous dysplasia.

Following this, X-rays were taken of the long bones and skull, but no evidence of other tumors was found. The mass from the left jaw was later removed, the pathological diagnosis being as above.

Fig. 1.

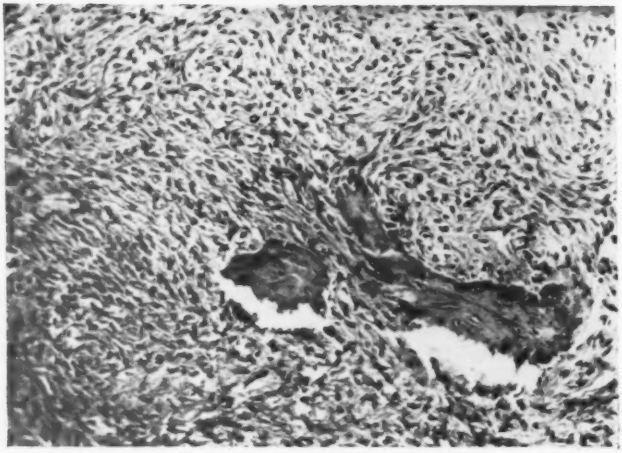

Fig. 2.

In November, 1950, this patient returned with a bony- painless swelling in the left side of the hard palate, of six months' duration. X-rays (see Fig. 3) show a tumor mass involving the left antrum with displacement of the nasoantral wall. Biopsy taken through left canine fossa. Pathological report: fibrous dysplasia.

Further X-rays were taken of the skull and long bone, in which the findings were negative. The serum calcium and phosphorous levels of the blood were negative; however, it is of marked interest that this man's sister was in the hospital six months previously for the removal of a parathyroid tumor with widespread skeletal involvement.

Operation November, 1950, by means of a left Caldwell-Luc approach, the growth was removed. The antrum was filled with what appeared to be fibrous tissue with a bony trabeculae which were almost like the cell partitions of a mastoid.

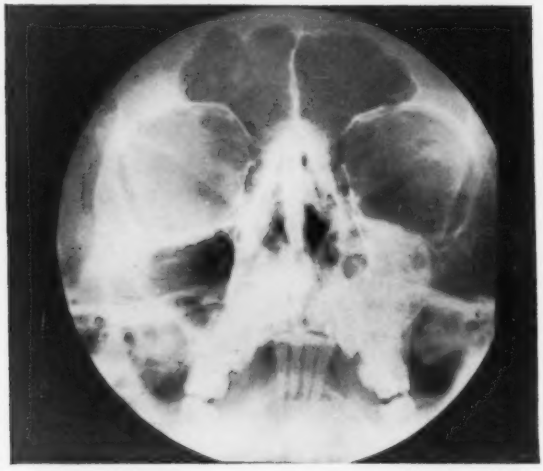

Fig. 3.

Case 2: A. C., a 15-year-old white female, gave a history of a painless lump, left parieto-occipital region of the skull, accidentally noticed 18 months previous to admission. No history of previous injury. X-ray of skull: evidence of wide area of bony change occupying the upper and midposterior part of the left parietal bone, about three inches in diameter, producing a considerable osseous reaction in regard to the outer plate. Findings suggest presence of a meningioma. Operation October, 1949. When skull comprising the tumor was lifted from the dura, there was no evidence of meningioma, but a rather soft granular tumor of the bone, bluish in color. Pathological diagnosis: fibrous dysplasia. Bone is present in trabeculae, between which are masses of fully formed adult fibrous tissue; there is no evidence of diploe, but there is evidence of proliferative and degenerative changes.

Case 3: J. S. A., a 21-year-old Japanese male. History of painless, gradually increasing swelling of the left cheek, 12 years. No history of injury. Examination reveals a hard mass involving anterior wall of left superior maxilla and extending into zygoma. The infraorbital ridge

appears to be pushed upwards, and the nasal bones appear to be pushed to the right; the hard palate on the left side is also involved. There was no exophthalmus or diplopia.

X-ray report (see Fig. 4): The left malar bone is increased in size. It is homogenously dense. The density extends laterally through the anterior half of the zygomatic arch, and medially it encroaches upon the lateral half of the left maxillary sinus. The medial portion of this sinus is aerated. The floor of the left orbit is elevated in its anterior rim. Biopsy revealed fibrous dysplasia.

Fig. 4.

April, 1951, mass removed under general anesthesia by left Mouré incision. Spongy tumor removed, involving left antrum, roof of orbit, anterior two-thirds of hard plate and anterior one-third of zygoma.

Microscopically, the section showed a dense network of wide bone trabeculae. The dense fibrous connective tissue fills the marrow cavities. All the bone is of a friable woven type, the bone cells being unevenly dispersed in the matrix through which fine fibrillar markings are distributed. This trabecular change extends right to surface of the lesion, there being no true cortex between it and the overlying muscle (see Fig. 5).

Case 4: N. P., a 44-year-old white female. Since 1932, and subsequent 14 years, has had a swelling of the right cheek. Physician made a diagnosis of acute maxillary sinusitis with operative drainage, 1935, but swelling never entirely subsided. Tumor did not progress in size until 1945, when it apparently involved the right zygomatic region and right

temple with periodic pain in the region of the right antrum, right temporal region and right ear. Admitted to Toronto General Hospital, 1946. X-rays of skull (see Fig. 6) and sinuses show a bony lesion involving right zygoma, maxilla, greater and lesser wings of sphenoid on the right

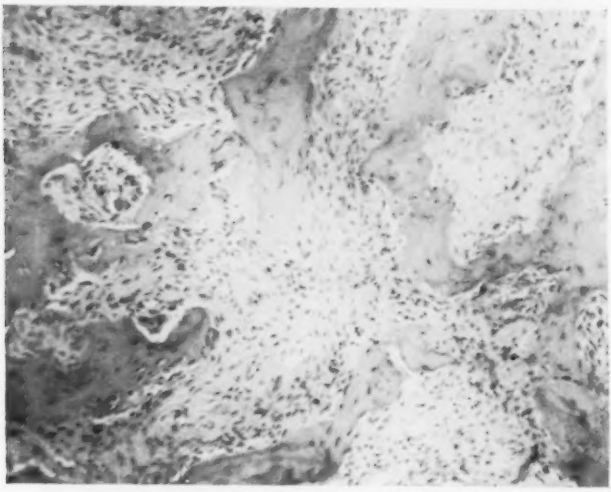

Fig. 5.

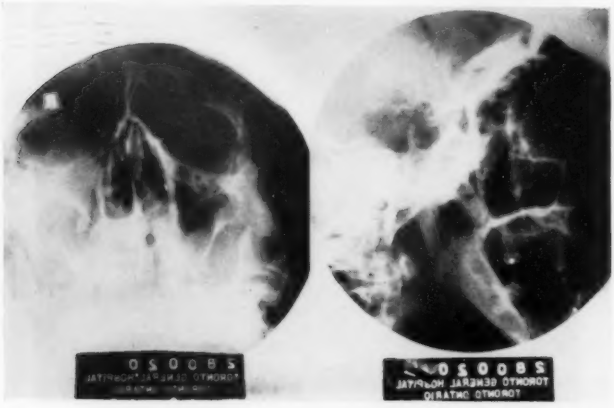

Fig. 6.

Fig. 7.

Fig. 8.

side, the squama of the right temporal bone and the supraorbital ridge of the right frontal bone. Biopsy taken through right canine fossa. Pathological diagnosis: fibrous dysplasia. Moderate cellular fibrous tissue, small masses of bone scattered through the fibrous tissue, a few blood vessels present. A considerable number of multinucleated osteoclast seen around the edges of many Haversian canals which are distended with fibrous tissue. X-ray therapy was carried out without any effect.

Case 5: D. W. A 43-year-old white female, married; mentality that of a high grade moron. One child six years old attending a school for the deaf. First seen in 1933 and gave a history of being pushed against a wall, injuring her face, at the age of 16 years. Examination reveals a hard, bony mass involving the left nasal bone, floor of the left orbital and left frontal bone, with marked prominence of the supraorbital margin. The space between the eyes is increased and the left eye is displaced downward (see Figs. 7 and 8).

There is no diplopia or proptosis. Examination of the nose reveals that the left middle turbinate is markedly enlarged and hard and has been pushed downwards almost to the floor of the nose—the ethmoid sinus has been obliterated.

Fig. 9.

There is an irregular cafe-au-lait pigmented area on the left buttock, 4 by 3 cm. The patient gives a history of menstruation at five years of age.

X-ray examination shows a well marked thickening (see Figs. 9 and 10) and increased density of the base of the skull. In the frontal bone, the diploic space is markedly widened and shows irregular areas of density and translucency. This process extends downward to produce a marked prominence of the supraorbital margin on both sdes and also extends medially to the orbit on the left side. The space between the orbital cavities is markedly increased.

Fig. 10.

X-rays were taken of the long bones and pelvis, which were negative. The serum calcium and phosphorous levels of the blood were negative.

Biopsies were taken from the frontal bone above the left eye and from the left middle turbinate. Pathological diagnosis: fibrous dysplasia (see Fig. 11), showing spicules of bone within whorls and strands of fibrous tissue, and a section from another area. Fig. 12 shows a mass of dense sclerotic bone.

Fig. 11.

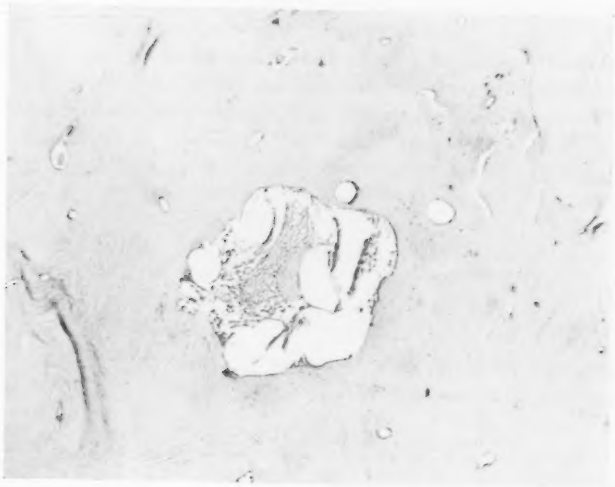

Fig. 12.

SUMMARY.

Five cases of fibrous dysplasia are reported; the correct diagnosis was made only on pathological report.

The lesions were asymptomatic except in one case, and in only one instance was it related to trauma.

In one of the male cases, a family history showed one sister having had a parathyroid tumor with widespread skeletal lesions. This case was also interesting in that he returned with a lesion in the antrum and hard palate 18 months after his original lesion had been removed.

Marked hyperostotic changes occurred in the skull in one instance; in this case pigmentation of the skin was present, and a history of precocious menstruation.

Most writers believe that the manifestations of the disease are congenital. There is no histological evidence suggesting that the disease is neoplastic in that mitosis or atypical cells are not present. Schlumberger feels that localized monostotic fibrous dysplasia is always a response to injury and, therefore, inflammatory in nature; Towson[12] reported a case of monostotic fibrous dysplasia of the mastoid bone in which there was a previous history of trauma, but other writers are at a loss as to whatever stimulates the continual activity of the undifferentiated fibrous tissue and most writers feel that the monostotic forms of the disease are a limited form of the polyostotic fibrous dysplasia. It is a chronic, slowly developing disease which manifests itself generally in childhood and persists throughout life but frequently is quiescent in the adult.

BIBLIOGRAPHY.

1. Von Recklinghausen, F. D.: *Festschrift*, Berlin, R. Virchow, 1891.

2. Morton, J. J.: *Arch. Surg.*, 4:552, 1922.

3. Elmslie, R. C.: *Brit. Jour. Surg.*, 2:17, 1841; *Proc. Roy. Soc. Med.*, 27:973, 1943; *St. Barth. Hosp. Report*, 68:147, 1935.

4. Winter, H.: *Zentralbl. f. Chir.*, 56:2647, 929.

5. Hunter, D., and Turnbull, H. M.: *Brit. Jour. Surg.*, 19:203, 1931.

6. Jaffe, H. L.: *Arch. Path.*, 16:63, 1933; *Ibid.*, 16:236, 1933; *Jour. Mt. Sinai Hosp.*, 12:364, 1945-1946; *Bull. N. Y. Acad. Med.*, 22:588, 1946.

7. Albright, F.; Butler, A. M.; Hampton, A. O., and Smith, P.: *N. Eng. Jour. Med.*, 216:727, 1937.

8. Lichenstein, L.: *Arch. Surg.*, 36:874, 1938.

9. Lichenstein, L., and Jaffe, H. L.: *Arch. Path.*, 33:777, 1942.

10. Duff, Murray and Pritchard: *Amer. Jour. Med. Sci.*, 222:313, 1951.

11. Schlumberger, H. G.: *Mil. Surg.*, 99:504, 1946.

12. Towson, C. E.: *Arch. Otolaryngol.*, 52:708, 1950.

702 Medical Arts Building.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES.

The 1952-1953 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1, 1952, and continue for a period of 10 months. Detailed information and application forms can be secured from Dr. William L. Benedict, executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn. Registrations should be completed before Aug. 15.

THE PROBLEM OF THE PRESCHOOL DEAF CHILD.

(Diagnostic Methods and the Otologist's Role in His Rehabilitation).*

JOHN E. BORDLEY, M.D. (by invitation),
Baltimore, Md.

Impairment of hearing in the preschool child poses two problems to the practicing otologist. First, he must have at his command the means necessary to determine the fact that the child has a hearing impairment. If possible, he should learn the type of hearing loss and the degree of that loss. Second, he must be able to help the parents plan for the child's future. Such planning should include not only the medical measures that might be indicated to improve the hearing but also a communicative training program for the child with the doctor acting as counselor. It is proper that the otologist assume this position as advisor because such a doctor-patient relationship has, through the years, proved most successful in keeping family cooperation during a long range training program. It is to the doctor that families in difficulties bring their children, and it is their faith in his ability to diagnose and advise that is the foundation stone on which the science of audiology should be built. To be useful in such a rôle, the otologist should have an understanding of the problem of language and speech training as well as those of the science of otology.

To make the diagnosis of hearing impairment in a group of preschool children is not easy. It is time consuming, frustrating and, in many instances, it results in the otologist's being more hopeful than sure that he is right. A diagnostic process in such a group of children should include a complete and detailed history which covers the family background,

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prenatal and birth history, etiological factors predisposing to hearing impairment, and the developmental pattern of the child. In many instances, such a history will reveal the necessity of psychiatric consultation to aid in evaluation. Included in any diagnostic procedure must be a careful physical examination of the ears, nose and throat. Finally, every possible available means should be employed to obtain accurate determinations of the child's hearing levels. All the data thus assembled must be assayed before any final diagnosis can be made.

Five hundred thirteen children under the age of six years have been examined during the past two and one-half years in the Hearing and Speech Clinic at Johns Hopkins Hospital. The median of this group lies in the third year. It might be well to review the practices evolved for the examination of these children and to discuss briefly some of the findings in this group. In order to make a comprehensive coverage of the patient's history, a questionnaire has been developed (see Fig. 1). This inquires into: *1.* The family background. *2.* The prenatal and birth history. *3.* The diseases, accidents and operations of early childhood. *4.* The general developmental history, including social maturity and hearing and speech progress. In general, we have found the mother the most satisfactory individual to interview when filling out these questionnaires. After the questions have been answered, the child receives a complete otological examination including inspection of the nose, nasopharynx and Eustachian orifices with the electric nasopharyngoscope. The child is then taken to the playroom where his performance with games and toys is observed. General performance tests are given, including some of the Gesell Development Series. Children with suspected mental retardation or suspected emotional disturbances are referred to the Children's Psychiatric Clinic for further evaluation. The hearing examination is divided into three general types: *1.* the tuning fork tests, which include both bone and air conduction; *2.* observations made while the child is in the soundproof room, introducing monitored sound effects, words and masking noises through a loud speaker.

FIG. 1.

PERTINENT FACTS FOR THE HISTORY OF A YOUNG CHILD WITH SUSPECTED HEARING LOSS.

Sex Birth Date Age Years Months

FAMILY HISTORY

1. Hearing impairment in family—age of onset in ancestors; 2. Age of siblings—list of any defects; 3. Miscarriages.

HISTORY OF PREGNANCY

1. Rh compatibility; 2. Illness during pregnancy (particular attention to infections by viral diseases and unexplained high fevers); 3. Immunization during pregnancy; 4. Bleeding; 5. Toxemia; 6. Premature delivery.

DELIVERY

1. Normal: long—short; 2. Induced: forceps—high—low; drugs.

POST-DELIVERY

1. Normal appearance of infant; 2. Abnormal appearance: anatomical deformities; paralysis; cyanosis; jaundice.

GENERAL HEALTH

1. Serious illness; convulsions; loss of consciousness; 2. Children's diseases—age; 3. Unexplained fevers—age; 4. Illness associated with immunization—age; 5. Serious injuries—age.

E N T HISTORY

1. Otitis media; 2. Mouth-breathing; 3. Tonsillitis; 4. Allergies; 5. Operations; 6. Frequently recurring colds.

DEVELOPMENT

1. General
   a. Age of onset of walking.
   b. Age of self-feeding.
   c. Age of toilet-training.
2. Speech and language
   a. Age of onset of speech.
   b. Normal babble; did it stop? when?
   c. When did child use two-word sentences?
   d. Is speech understandable?
   e. Does child understand without gesture?
   f. Is vocabulary-development reasonable?
3. Hearing
   a. Response to noises in local environment.
   b. Response to conversation; to calling—what distance?
   c. Attention paid to radio—to T.V.
   d. Other indications of awareness of sound.
4. Social maturity
   a. Self-management of clothes and toilet habits.
   b. What are typical play-activities?
   c. How adjusted toward other children?
   d. Temper tantrums?
5. Education or group-experience
   a. Level.
   b. Progress.

When the child has language, object identification is tried, using monitored voice. No attempt is made at this time to obtain any unilateral hearing evaluations. *3.* Audiometry. This consists of both psychogalvanic skin-resistance audiometry[1] and pure-tone subjective audiometry. All of the 513 children under discussion have been examined with psychogalvanic skin-resistance audiometry. Where the cooperation of the child made it feasible, subjective audiometry has been attempted.

Psychogalvanic skin-resistance audiometry is well described in two recent paragraphs written by Dr. W. G. Hardy and Dr. Stacy Guild:[2] "By means of measuring changes in sweat gland activity it is possible to determine thresholds for pure tones, even in very young children. After a child, or an older person, is given a mild faradic shock a few seconds following a test-tone, and the experience is repeated five to 10 times, increased sweating occurs before the shock is given. This physiologic fact is the basis for the test-method known as psychogalvanic skin-resistance (PGSR) audiometry. Once this conditioning is established, the shock is omitted and the test-tones are presented as in standard audiometry, from which the method differs only in the manner of observing the patient's responses. Instead of raising a finger or pushing a button, the patient sweats if the tone is heard, and the presence or absence of this response is recorded by an ink writer on a roll of paper. Precisely what causes the sweating is unknown. This phenomenon has been known, however, since Fere's[3] work in 1888, and has been widely used since in many different kinds of psychobiologic measurements."

An analysis of the etiological factors underlying the hearing impairment in 485 of the 513 children examined in the Hearing and Speech Clinic can only be briefly scanned today. The 485 children are those with a known hearing loss. Twenty-eight of the children examined in the group under discussion had no evidence of hearing loss when studied by the above methods (see Fig. 2). In this analysis, 28 categories have been established. Some of them, such as cerebral degeneration, are rather loose and may later have to be broken down.

FIG. 2.

TOTAL NUMBER OF CHILDREN UNDER SIX YEARS OF AGE—513.

NORMAL HEARING, 28.

IMPAIRED HEARING, 485.

Analysis of Etiologic Factors Underlying Impaired Hearing of 485 Children.

Etiologic Factor	Number Children
Familial	18
Erythroblastosis (Rh Incompatibility)	29
Prenatal Rubella	24
Prenatal Hyperpyrexia	2
Prenatal Immunization	4
Prenatal Diabetes	1
Toxemia of Pregnancy	5
Anatomical Developmental Anomalies	16
Birth Injuries	13
Cyanosis at Birth	13
Cerebral Degeneration	26
Cerebral Palsy (No Known Rh Incompatibility)	32
Infant Diarrhea	1
Meningitis	43
Measles (Rubeola)	31
Whooping Cough (Pertussis)	8
Chicken Pox (Varicella)	6
Mumps (Epidemic Parotitis)	5
Scarlet Fever	2
Pneumonia	4
Jaundice Postnasal	3
Typhoid Fever	1
Poliomyelitis	1
Unexplained Hyperpyrexia	37
Neurotropic Chemicals. *1.* Lead. *2.* Citronella	2
Streptomycin	1
Middle Ear Lesions (Not Anatomical Anomalies)	17
Cause Undetermined	140

The largest single group is that in which no specific etiological cause could be determined. This includes quite a few adopted children whose past histories were unknown, so that complete data was unobtainable. The two classifications that have been of particular interest to us include the four children

born of mothers who received immune globulin during the first trimester of their pregnancy and the cyanotic group. The four women immunized during the first trimester of their pregnancy showed no evidence of measles, and it is quite questionable just how severely exposed three of them were. The fourth was definitely not exposed to measles, but was given immune globulin after one of her children developed poison ivy, which apparently was incorrectly diagnosed on the first day and later never resembled measles. The findings in these four children lead us to believe that some of the present work being done on prenatal immunization in some of the obstetrical and pediatric clinics should be assessed with the greatest care. Of the cyanotic group, all 13 of these children required prolonged oxygen therapy. Reviewing the picture as a whole, prenatal rubella, erythroblastosis and meningitis are still among the greatest contributors to the problem that faces us today.

Those people working on the problems of etiological factors predisposing to deafness will, undoubtedly, feel that the foregoing classification is not definitive. It was created only as a skeletal framework which can be added to or changed as our knowledge accumulates; in the Hearing and Speech Clinic, it has been modified considerably in the past three years.

Of the 513 children passing through the clinic, when the 28 normals had been excluded, the remaining children have an average mean "speech potential" of 57.8 db. Such "speech potential" is arrived at by using the average loss in decibels for the pure-tones 500, 1,000 and 2,000 cycles in the better ear of the examinee. The figure 57.8 db is better than one might expect in such a group. It is important because it means that children having such an average loss would have a fair prognosis for training with amplification. Useful amplification means that the child will have an advantage in speech development. Early discovery of such a hearing loss which can be helped by amplification means that the child has an opportunity to develop usable speech during his early years.

Such an acquisition of speech very frequently means that, with special courses to improve the communicative function, a child may be admitted to a normal educational program.

The careful evolution of an accurate diagnostic process is necessary if eventually we are to learn the degree and cause of the child's hearing handicap. Such a determination is only a part of the responsibility of the otologist. All of his findings must be interpreted to the parents. The degree of hearing loss, the type of loss, and the cause of that loss are all very important and necessary information to them. They should be told if anything can be done medically to help their child. The family must learn what their child's handicap will mean to the interrelationship in their family group. They should understand what their child's hearing impairment has contributed to its past development, especially when that development has seemed deviant from that of a normal pattern. They should realize what this handicap may mean in the child's future social adjustment. Such insight is necessary to both the doctor and the family in working out the pattern for the child's future education. It is such a program, set up early and intelligently, that so often means the difference between a normal and an abnormal upbringing. Early study and comprehension of the child's hearing loss offers our best chance to overcome the handicaps inherent in such a disability.

BIBLIOGRAPHY.

1. Bordley, John E., and Hardy, William G.: A Study in Objective Audiometry with the Use of a Psychogalvanometric Response. *Ann. Otol., Rhinol. and Laryngol.*, 58:3:751, Sept., 1949.

2. Item written for inclusion in the Manual for the Management of Preschool Children with Hearing Loss, to be published by the Committee on the Conservation of Hearing, of the American Academy of Ophthalmology and Otolaryngology.

3. Fere, C.: Note sur des Modifications de la Résistance Electrique sous l'Influences des Excitations Sensorielles et des Emotions. *C. R. Soc. Biol.*, Paris, 5:217, 1888.

RECRUITMENT AND SPEECH DISCRIMINATION LOSS.*

HENK C. HUIZING, Ph.D., and J. A. REYNTJES, M.D.,
Groningen, Netherlands.

The important rôle of the recruitment factor for the comprehension of speech in certain cases of perception deafness has been emphasized previously by one of us.[1,2] On account of theoretical considerations,[2] *a priori* was to be expected that a rather critical intensity level for optimum speech intelligibility combined with a critical distance for optimum speech understanding capacity for conversational voice might exist in connection with the recruitment phenomenon.

The occurrence of such cases was recently stressed again by the less satisfactory application of electronic aids on hard of hearing individuals, whose loudness loss presents a typical regressive character for stimuli of gradually increasing intensity and also by the less successful results of auditory training in young children (*cf.* 2, item 11) with the same type of hearing impairment following meningitis, streptomycine, etc. This last point has been confirmed recently by the work of Wedenberg.[3] An explanation[2] of these phenomena can be found in the subjective distortion engendered by this type of pathological loudness perception (pathological subjective distortion).

For the purpose of a better insight into this problem a series of experiments was set up, which are described *in toto* in a thesis by one of us,[4] and in which case reports also are given. The principle of speech audiometry by applying the Groningen P. B.-lists to a high quality sound reproducing system with a uniform frequency characteristic and a linear intensity response circuit was used.

*From the Audiology Department of the University Ear Clinic, Groningen, Netherlands.

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Two types of articulation curves have previously been differentiated by Davis[5] and Silverman[6] (see C-type and P-type in Fig. 1). The experiments revealed a third type, which could be related to the recruitment phenomenon (see R-type, Fig. 1).

1. C-type—pure sensitivity loss, which means that the hearing function for speech may be restored to normal (N) merely by adequate amplification. This type is typical for conduction deafness. In these cases the articulation curve is simply shifted over a distance corresponding to the average threshold loss for pure tones in the speech region.

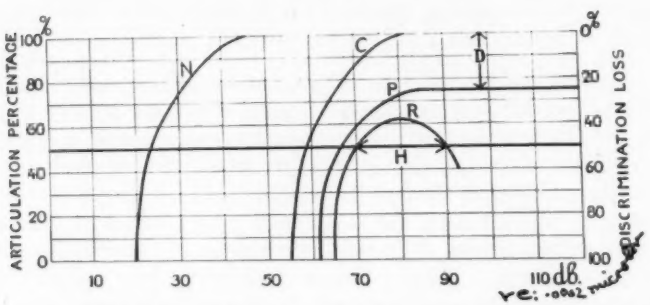

Fig. 1. Articulation curves. (N) Normal ear. (C) Pure conductive impairment. (P) Perceptive impairment with constant value of (D). (R) Perceptive impairment with variable value of (D), related to recruitment. (H) Half-way width.

2. P-type—sensitivity loss combined with loss of discrimination (D) caused by a perceptive type of hearing loss. In such a case the articulation percentage does not pass a certain maximum value; after a steep rise the articulation curve merges into a horizontal line, the so-called saturation level. The analysis of speech is now restricted to a limited number of speech elements. From this we can understand that the operational range of the aided ear is still confined to a limited distance, depending upon this saturation level. It is also obvious why speech reading is very valuable in these cases because of the combined effect of visual and acoustical clues.

3. R-type—sensitivity loss combined with loss of discrimination by preponderating subjective distortion due to a pathological loudness function (recruitment).

This type of articulation curve shows a typical optimum region of intelligibility. When the optimum listening level (80 db in Fig. 1) is passed, a rapid fall of articulation score is found. The recruitment phenomenon causes a steep rise of the loudness perception of the proffered speech as soon as the threshold of detection is passed. The articulation score, however, does not keep pace with this loudness sensation, inasmuch as the curve presents a resonance-hump, which may be more or less broad. At high intensity levels this results in a discrepancy between the loudness of speech and its intelligibility.

The area of the "helmet-shaped" upper part of this curve—*chapeau de gendarme*—with the 50 per cent level as a borderline, determines the social adequacy index[5] of the impaired ear. When fitting a hearing aid, the "halfway-width" (H) of the curve—the baseline of the helmet—and also its height, should be made as great as possible by auditory training before making the final choice. The figure H is as important as D, since H determines the width of the area over which a hearing aid user can manage with his instrument by a certain setting of its volume control.

This R-type of articulation curve should not be confused with the same type of bent curve mentioned in literature,[7] which apparently is connected with *objective* distortion. It is well known that amplification circuits as used in conventional hearing aids provoke a certain amount of harmonic distortion which may involve even a large discrimination loss for the aided ear when the circuit load is gradually increased. Consequently, an artificial discrimination loss is introduced, which then appears also in cases of pure conduction deafness,[7] whereas the same ear would not show such a loss if high quality laboratory equipment had been used for the speech tests.

Even in the normal ear the articulation curve represents a downward trend at very high intensity levels when standard laboratory equipment is used. This small discrimination loss can be considered as a normal subjective distortion on a pure *physiological* basis which may be neglected in comparison with the *pathological* subjective distortion caused by the recruitment symptom.

Although the R-type might be considered as a special case of the general P-type, it has been split up because of the predominating influence which the speech intensity exerts on the value of D. This is specific for recruitment.

The regular P-type with its saturation effect presents a *constant* value of D, which may have various causes:

a. Complete drop-out of a certain part of the frequency scale (dip or abrupt threshold-audiogram).

b. Subjective masking (sloping type of audiogram, tinnitus). In a sloping type of audiogram the low components of speech prevail over the weak high tones, resulting in an obliteration of the treble part. The voiceless consonants are always masked in these cases and as the slope of the curve increases, a larger part of the gamma will be involved.

In the normal ear the *center of intelligibility* is at about 1,500 cycles per second. In a sloping audiogram this center dips more and more as the slope of the curve increases. This means that the serviceable part of tonal range is limited to a more or less narrow region in the bass. Consequently, the value of D increases.

c. Tonal dispersion and fuzziness by partially delayed[8] analysis (presbyacusis) or by asymmetrical analysis (diplacusis).

In many cases the above mentioned *constant* types of discrimination loss will be mixed with the *variable* R-type. The occurrence of this type of articulation curve in connection with recruitment has been confirmed recently by Azzi.[9]

It has been advocated that peak-clipping and automatic volume control circuits are apt to compensate for recruitment phenomena. This is true so far as concerns the prevention of uncomfortable loudness levels, for which these circuits operate as a vent; however, they do not counter-balance subjective distortion. Peak-clipping has no substantial influence on the articulation score of a normal ear, although it involves a certain amount of objective distortion. Combined with subjective distortion, however, intermodulation effects may enhance considerably the value of D at the right part of the helmet-shaped curve.

The existence of the variable type of discrimination loss has been connected to speech intensity up to now, but naturally the change of D, *i.e.*, the shape of the curve, will depend also upon the type of frequency response characteristic used. This is especially true when the subjective distortion originates from a somewhat narrow band of the tone scala. If such a band can be suppressed, the variable type of curve may change into a constant type in which the saturation level exceeds the optimum level of the original. This would increase the practical value of a hearing aid.

Although recruitment is still a complicated matter, *band-clipping*—*c.q.*, partial suppression—seems to be an advantageous procedure for certain cases with a more or less flat threshold audiogram, that still covers the complete gamut. Then the frequency response characteristic should be chosen so that the center of intelligibility is moved to a part of the frequency scale, in which the subjective distortion is as low as possible, while the contributing band-width still remains large enough to obtain better conditions.

The data on 15 cases of the R-type using uniform and linear amplification present an intensity-spread from 70 db to 90 db for the optimum listening-level. The average of about 80 db may provide a useful figure for an adequate volume control setting for auditory training of young children whose hearing impairment is characterized by the recruitment phenomenon; furthermore, it seems advisable to recommend an intensity

response characteristic with a stabilized output. Such children would stay behind in speech development if the gain of their hearing aids would be raised to very high levels as is necessary in cases without recruitment. To a certain extent the poor results of auditory training in these children may be explained by the fact that the recognition of the proffered speech elements depends greatly upon the rather critical value of the speech intensity level.

It may be concluded that in all cases of perception deafness more information about the loudness function of the organ should be collected.

SUMMARY.

Speech audiometry using Groningen P. B.-lists presents three types of articulation curves (C-type, P-type and R-type). The R-type, characterized by a kind of resonance-hump, can be related to the subjective distortion caused by recruitment.

The variable type of discrimination loss embodied in this R-type can be defined by the discrimination loss D and the halfway-width H. In connection with the constant type of discrimination loss (P-type) three kinds of hearing impairment are considered.

The influence of objective and subjective distortion on the articulation curve is discussed. Subjective distortion may be of pure physiological as well as of pathological origin.

Peak-clipping and automatic volume control circuits do not compensate for subjective distortion. By means of band-clipping the center of intelligibility can be moved sometimes to a part of the frequency scale in which the subjective distortion can be neglected.

The average optimum listening level of 80 db may provide a useful figure for auditory training of young children whose impairment is characterized by the recruitment phenomenon.

REFERENCES.

1. Huizing, H. C.: *Acta Oto-Laryngol.*, 36:346, 1949.

2. Huizing, H. C.: *Acta Oto-Laryngol.*, Suppl., 1952. *Proc. Intern. Course of Audiology*, Sept., 1950.

3. Wedenberg, Erik: *Acta Oto-Laryngol.*, Suppl. 94, 1951.

4. Reyntjes, J. A.: Spraakaudiometrie. Thesis, Groningen University, Feb., 1951 (in Dutch).

Huizing, H. C., and Reyntjes, J. A.: Proc. Coll. O. R. L. A. S., Rome, Sept., 1950; *Rev. Laryngol., Otol. and Rhinol.*, Supp., p. 215, May, 1951.

5. Davis, Hallowell: The Laryngoscope, 58:761, 1948.

6. Silverman, S. R.: *Arch. Oto-laryngol.*, 51:786, 1950; *Jour. Laryngol. and Otol.*, 64:23, 1950.

7. Hudgins, C. V., *et al.*: *Jour. Ac. Soc. Amer.*, 20:241, 1948.

8. Flanagan, J. L.: *Jour. Ac. Soc. Amer.*, 23:303, 1951.

9. Azzi, A.: *Riv. Audiol. Prat.*, Maggio, p. 44, 1951.

182 Oostersingel.

1952 SUMMER MEETING, VOLTA SPEECH ASSOCIATION FOR THE DEAF.

The 1952 Summer meeting of the Volta Speech Association for the Deaf will be held in New England, June 16-20, 1952. Theme: "That the Deaf May Speak."

The program will begin on June 16 in Northampton, Mass., when the Clarke School will welcome our Association members with a series of demonstrations. The Clarke program will end Tuesday at noon and the opening session of the main convention will be held Tuesday evening, June 17, at the Hotel Kenmore, 490 Commonwealth Avenue, Boston, at 8 p.m.

The program will continue Wednesday, Thursday and Friday at the Horace Mann School and the Hotel Kenmore.

Members may register either in Northampton, Mass., or Boston.

Nathan P. Harris, Program Chairman.

DESTRUCTIVE CARCINOMA OF THE NOSE FOLLOWING A STAB WOUND.

ALBERT B. SELTZER, M.D.,
Philadelphia, Pa.

Malignant growths of the external nose are very deforming unless they are treated by early eradication. Since the epidermoid cancers, with which this paper is concerned, are slow-growing and run their early course as shallow, gradually-spreading ulcers, they are often wrongly diagnosed and neglected. They occur wherever there is transitional or squamous epithelium, both externally and in mucous membranes.

Such malignant growths of the skin were formerly known as epitheliomas. They are often caused by irritation or injury, particularly if the former is of moderate degree and frequently repeated. The growths first appear as a small, focal thickening of the skin, which may become nodular, and which breaks down at the center with ulceration, or they may also develop in the margin of chronic ulcers, that in the beginning show no sign of malignancy. The malignancy is indicated by their tendency to infiltrate the surrounding tissue and to spread by metastasis.

Epidermoid malignancy occurs with about equal frequency in both sexes. The bodily changes that occur with ageing appear to be a factor in their occurrence, since they arise more often during the involutional period, from 45 years onward. Exceptionally, they may occur during early life.

Epidermoid carcinoma must be differentiated from dermoid cyst, which it may sometimes resemble, but which is a benign process. The latter formation may be congenital, or it may be a result of a penetrating injury, in which a small group of cells from the germinative layer of the skin is embedded in

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the underlying tissue. This fragment of skin tissue thus becomes more or less analogous to a tissue culture. The buried cells continue to multiply, and since they are closed in below the skin surface, the cells which normally form the cuticle accumulate with a resulting cyst formation. There is no tendency to infiltration or to spreading by metastasis; therefore, they are not malignant.

In the absence of injury, the skin of the nose and face are frequently sites of new growths, both benign and malignant. They may start as simple keratoses in the elderly and go on to later ulceration if infiltrated. Others may appear as a small nodule in the skin, which in time becomes umbilicated and later breaks down, forming a chronic ulcer. Only when these ulcerating skin areas begin to increase in size and abnormal cells start to infiltrate the surrounding tissue is malignancy demonstrable.

The case considered here is that of a man of 75 years of age. He has been a prison guard for many years, and 20 years ago he was stabbed in the nose. The long delay in the appearance of definite malignancy, together with the nature of the injury, make this case of more than usual interest.

Before he received the stab wound, about 20 years ago, there had been no abnormality of the skin of the nose, as far as the patient is aware. After this injury, the wound would partly close, only to break down again. The granulating area would become covered with a scab, but when the scab separated and was shed, the underlying surface became ulcerated once more. Thus, an alternating of a feeble attempt of surface restoration and open ulceration has been going on for many years, with frequent episodes of bleeding from the nose.

Together with the chronic ulceration of the nasal dorsum, there developed a moderate enlargement of the lower segment of the nose, of the nature of a rhinophyma. Just when this latter condition began is not altogether certain, but the patient is sure that there was nothing of this sort evident before the injury and the development of ulceration.

Since the injury, there has been treatment from time to time, at various clinics, with the use of a variety of external applications and there is also a history of a few X-ray treatments, of which no further information has been obtainable. A prothesis to mask the ulcerated area was worn at one time for a while, but it was found to be very awkward and difficult to keep in position, so it was abandoned.

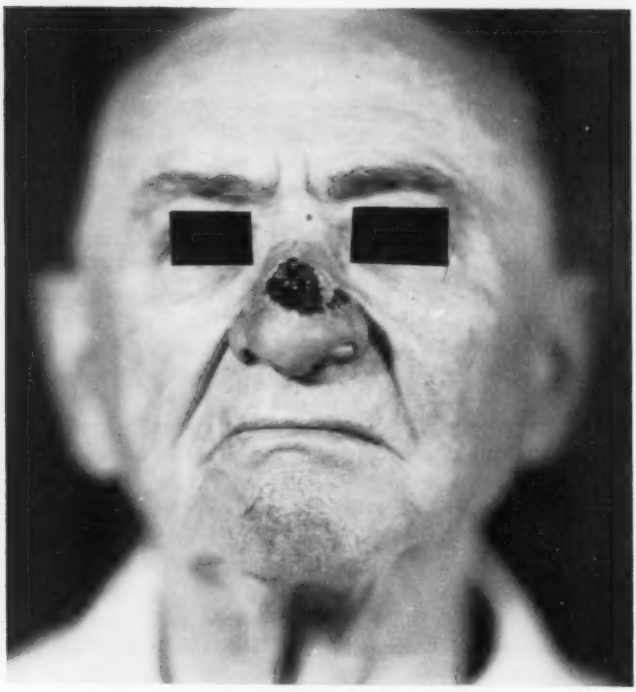

Fig. 1A.

When the patient was first examined by the writer recently, the nose was enlarged, and over the junction of the bony and cartilaginous septums was an open ulcerating area of irregu-

lar outline, roughly 1 cm. in its anteroposterior diameter, by 2.5 cm. laterally, extending well down over the left side of the nose and encroaching upon the ala (see Figs. 1A and 1B).

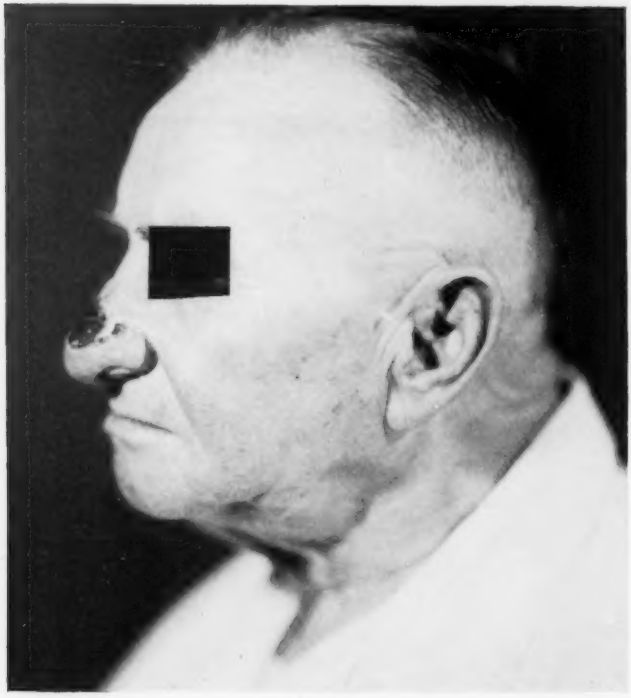

Fig. 1B.

A biopsy specimen was taken, the report on which confirmed the clinical diagnosis of epidermoid carcinoma (epithelioma). The histological report specified that the specimen consisted of fibrous tissue, extending downward into which were a number of rounded islands of epidermoid cells, and in some of these cell masses there were characteristic keratin pearls.

An X-ray examination was also made, with a report of normal appearing nasal and facial bones. The frontal, ethmoid, sphenoid and right maxillary sinuses were all clear, but there was opacity of the entire left antrum. Although the Roentgenologist considered that this condition was probably due to a chronic sinusitis, because of the diagnosis of the nasal ulcer as malignant, a Caldwell-Luc operation was performed upon the left maxillary sinus. The tissue was sub-

Fig. 2A.

mitted for a histological examination, and the report received gave the gross report of the specimen as small, thin, gray-white friable fragments of slightly roughened mucosa, and

four rounded pieces of bone, about 5 mm. in diameter, covered partly by mucosa. The report on both these specimens was entirely negative for malignant changes both in soft tissues and in bone.

An operation upon the nose was then planned, and under general anesthesia (intravenous sodium pentothal) an extensive incision of the nose was made, with the electric cautery,

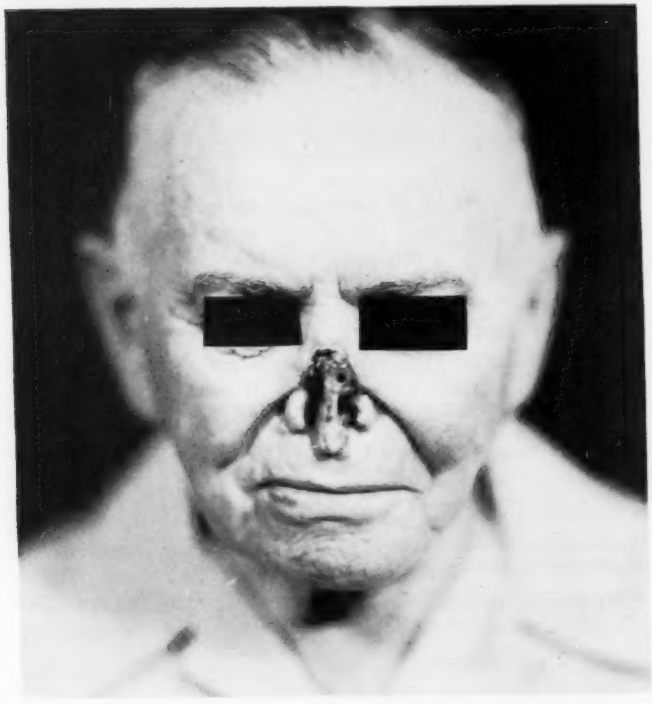

Fig. 2B.

including the entire tip, but excluding the alae, and reaching well up over the bridge, to include all the malignant process (see Figs. 2A and 2B). Bleeding was profuse but controllable.

The excised specimen was sent for pathological examination, and was reported to have a sufficiently wide, malignant-free border.

The patient made an excellent recovery, with the loss of only two weeks of working time. After the operative wound is entirely healed, a period of six months or more, a nasal reconstruction will be done, using a forehead flap. Meantime, a bandage is worn over the defect in the nose, and is proving quite satisfactory.

SUMMARY.

A case is described of a prison guard, who received a stab wound of the nose 20 years ago, which was followed only recently by the development of rhinophyma and malignant new growth.

The lower segment of the nose has now been amputated, to include all abnormal tissue, and a reconstruction will be done after healing is complete, using a forehead flap.

BIBLIOGRAPHY.

ANDERSON, W. A. D.: Pathology. St. Louis, Mo.: The C. V. Mosby Co., 1948.

BOYD, W.: Textbook of Pathology. 5th Ed. Philadelphia, Pa.: Lea and Febiger, 1947.

EWING, J.: Neoplastic Diseases. 4th Ed. Philadelphia, Pa.: Saunders, 1940.

FOOT, N. C. Identification of Tumors. Philadelphia, Pa.: Lippincott, 1948.

NICHOLSON, G. W.: Studies on Tumor Formation. St. Louis, Mo.: The C. V. Mosby Co., 1950.

2104 Spruce Street.

THE TREATMENT OF NASAL POLYP BY THE RUSKIN METHOD.*

JUAN JIMENEZ CERVANTES, M.D.,
Murcia, Spain.

In THE LARYNGOSCOPE (Vol. LX, No. 1, 1950) we find a report published by Ruskin on the treatment of nasal polyp by means of intrasinual injections or instillations of penicillin-procaine.

Ruskin used this treatment in an almost fatal recurrence of polyp after its surgical removal, although, as Halle and Hajek advocate, the extirpation was done radically and the ethmoid totally emptied. Later, this treatment was modified as regarding the influence of allergy on the production of polyp was recognized.

The nasal polyp is described as a continuation of the sinus mucosa grossly distended by the accumulation of transudate. The larger production of the latter in comparison with its reabsorption speed gives rise to its continuous growth; therefore, the keystone of the treatment is this: to increase reabsorption rather than to promote transudate. In order to attain this condition, reduction of the allergic activity and control of infection must be obtained.

We will point out briefly the fundamental management: Penicillin-procaine salt is stable, and does not produce hydrolysis. Due to the small absorption capacity of the sinus mucosa, an action of deposit is produced within the sinus, so that the anti-infectious action is at its maximum. Another very important factor is the antihistaminic action of procaine.

Ruskin used anti-infectious and antihistaminic treatment in 60 cases of nasal polyposis with excellent success. He

*Read at the First Congress of the Spanish Society of Otology, Rhinology and Laryngology, 1951.

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introduced penicillin-procaine into the sinus by means of a modified Yankauer cannula on three consecutive days, then on three alternate days, and finally once a week for two weeks, the nose remaining free from polyps.

The description of this simple and highly successful procedure with its seemingly logical basis led us to use this treatment on eight patients (six adults and two children) and the result was absolutely nil. I lowered and increased the concentrations with the same result. I do not believe the failure to be due to my introduction of the drug by puncture, because by this means and by injecting penicillin alone it was possible to cure a patient suffering from a purulent maxillar sinusitis accompanied by a single polypoid formation, of brilliant red color, similar to a huge inflammatory edema, as usually seen on the uvula or the posterior pillar in peritonsillar abscesses or in the arytenoids in some forms of laryngeal tuberculosis. This polypoid formation disappeared at the first injection of penicillin into the sinus, and the sinusitis (monosinusitis, which precludes allergy) was cured after four more punctures. There has been no recurrence in three years.

CONCLUSIONS.

From our experience in the treatment of eight cases of nasal polyposis, we conclude:

1. The injections of penicillin-procaine into the sinus do not cause the disappearance of the polyps in the middle meatus, when it is a true polyp.

2. Amelioration and even disappearance of inflammatory edema localized in the middle meatus, and following a more or less acute sinusitis, is possible.

3. This was deduced from the results obtained by injecting antibiotics in the treatment of sinusitis. In those cases with a strong chronic hyperplasis of the mucosa the utmost result was sometimes temporary sterilization, while in the acute or subacute cases with medium or even strongly increased mucosa, the return to normality is immediate.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

May 1, 1952.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chcago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2.
Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model 103BM.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model J-92.
Manufacturer: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.
Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.
Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Meeting:

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Meeting:

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Meeting: Palmer House, Chicago, Ill., Oct. 12-17, 1952.

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Meeting: Palmer House, Chicago, Ill., Oct. 6-10, 1952.

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Place: Army and Navy Club, Washington, D. C.

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The Vancouver Hotel, Vancouver, B. C., Monday, Tuesday, Wednesday, March 31, April 1, 2.

The Pioneer Hotel, Tucson, Ariz., Monday, Tuesday, Wednesday, April 7, 8, 9.

Royal York Hotel, Toronto, Canada, Thursday, Friday, Saturday, May 15, 16, 17.

Those who plan to attend a Sectional Meeting may select the meeting which in time or place is most convenient.

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Meeting: Palmer House, Chicago, Ill., Oct. 17, 1952.

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Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: January, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

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Time and Place: Caracas, Venezuela, 1953.

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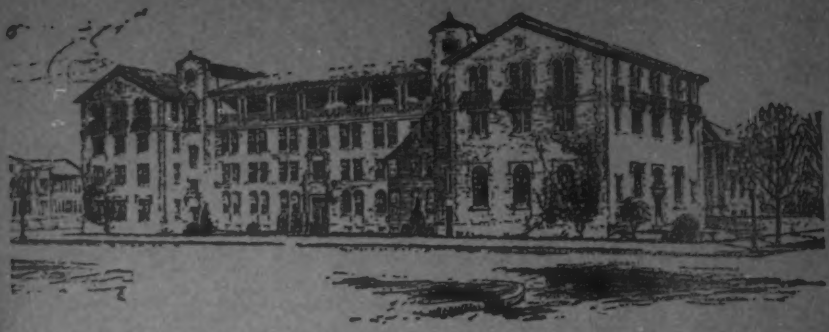

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